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Table of Contents.

THE PATHOGENESIS AND NATURE OF DIFFUSE COLLOID GOITRE IN AUSTRALIA.

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DIFFUSE enlargement of the thyroid gland without clinical signs of increased secretion of hormone may be encountered in any locality. In some places the condition is more common than in others and is called endemic goitre. In Australia and elsewhere throughout the world such thyroid glands are enlarged mainly because they contain excessive amounts of colloid. These glands and similar ones seen sporadically are included in the term "diffuse colloid goitre". In an area where goitre is endemic many, or even most, of the enlarged glands are nodular. It is considered that these have passed through the stage of diffuse colloid goitre (Taylor, 1956; Means, 1948). Almost all simple goitres become nodular with the passage of time.

The Normal Thyroid.

The normal thyroid gland is a very labile organ, the structure of which varies with age, geographic locality and the different stimuli met in normal daily life. I can find no published survey of the anatomy of normal thyroids in Australia. The following brief account is based mainly on my own observations and on the study of tissue provided by colleagues.

The thyroid gland first appears in early fœtal life. By the third month it contains thyroxine (McGirr and Hutchison, 1955). At birth the vesicles are small and are lined by tall cells. The walls are folded, and there is little colloid and a high proportion of connective-tissue stroma. As the child grows the gland grows at about the same rate. The vesicles increase in size, mainly by dilatation of the lumen. The lining cells multiply and become flatter, but probably do not decrease in volume (Figures I and II). At puberty the gland grows rapidly. This is explained as possibly due to increased metabolic rate of the body or to a general increase in the activity of the anterior lobe of the pituitary gland (Hubble, 1956). The normal gland at this stage is not visibly enlarged (Clements, 1948).

After puberty the thyroid gland resumes its steady rate of growth. The cells are now cuboidal or flatter, and the lumen of the vesicle is still enlarging. There is a greater variation in the size of vesicles than before (Figure III). At this stage various stimuli, such as severe emotional stress, cold weather (Uotila, 1939), pregnancy and general infections (Womack and Cole, 1929) may be encountered. These stimuli lead to temporarily increased size of the thyroid cells and to increased secretion of hormone. The cells become taller and may even form papillary projections into the lumen of the gland. The colloid is partly absorbed, and in histological sections spaces appear at its edges. These are probably the result of a proteolytic enzyme which is known to liquefy the normally gelatinous colloid before it can be absorbed (Michel, 1956). The liquefied colloid is frequently washed out or dissolved during the preparation of the section. When the temporary stimulus ceases, the gland usually returns to normal. However, the return may not be uniform. Some small areas often remain active and later pass to a stage of excessive colloid storage, forming obvious nodules. As would be expected, nodules are most frequently found in older people. In adult life the thyroid thus becomes variable in appearance, difficult to describe and to interpret.

After middle age the activity of the gland decreases as the metabolic rate of the body is reduced. In North America the gland has been shown to decrease in weight after the age of about forty years (Rice, 1938); but it is not known whether this is so in Australia. The vesicles now vary greatly in size. The greater part of the gland is occupied by distended vesicles lined by flattened cells; there are also many very small follicles (Figure IV). Rice (1938), in his study of normal thyroid glands in America, stresses the presence of the small follicles, and states that the gland may resemble that of infancy except for more variation in size of follicles. It is not clear whether there is a significant difference in the two countries. Nodules are common and often include colloid cysts, hæmorrhage, necrosis and fibrous scars.

From these normal changes in the thyroid gland some deductions can be drawn. A short stimulus to increased production of hormone leads to enlargement of the thyroid cells and absorption of the stored colloid. A steady prolonged stimulus, as occurs during growth, leads to multiplication of the cells. On the other hand a prolonged lowering of thyroxine requirements, as in old age, leads to the storage of large quantities of colloid in the gland. David Marine (1935) in America considered that much of the disease of thyroid glands represented pathological variations of these three phenomena.

Diffuse Colloid Goitre.

General Features.

Goitre is endemic in many countries. In Australia it is found in a wide area near the east coast of New South Wales and Victoria, and in many parts of Tasmania (Sutton, 1927). The affected areas of the world probably all have low iodine levels in water and soil. In addition there are a number of "goitrogens" which, taken in the food or water, will produce a goitre when the iodine intake is only just sufficient. These goitrogens have been known for many years, but their importance has become more apparent recently (Taylor, 1956). Clements (1955) has shown that they are transmitted to children by cow's milk in Tasmania.

The sporadic cases of diffuse colloid goitre are attributed to inefficient thyroid glands, a factor which may be inherited (Hubble, 1956; Webster and Chesney, 1930), and to peculiarities of the diet. Clements (1948) found in a survey of schoolgirls in two suburbs of Sydney that 6% of girls aged between twelve and fourteen years had visibly enlarged thyroid glands. He considered these to be examples of sporadic goitre. Most of the glands would probably revert to normal size in the next few years.

Types of Endemic Goitre.

The pathology of endemic goitre in Tasmania has been briefly discussed by Stuart (1955). Dr. M. A. Bundock, of Tamworth, New South Wales, has described in a personal communication his experience in this area, which has for Australia a high rate of endemic goitre. From these accounts it appears that the changes are the same as those found in North America about the Great Lakes and the Saint Lawrence River basin (Hellwig, 1932), and also in England and low-lying parts of central Europe (McCarrison, 1937). In the Alps and Himalayas some goitres are of a similar colloid type, but in the more severely affected areas many of the goitres are parenchymatous. In these localities there are a number of cretins, subjects with congenital goitre and people showing signs of hypothyroidism (McCarrison, 1937).

Pathology.

In developed colloid goitre the gland is swollen by enlargement of its vesicles, which contain excessive amounts of colloid. This may contain less iodine than normal. The vesicles, which vary greatly in size, are lined by flattened cells (Figure V). With careful searching small groups of tall cells may be found projecting into the lumen (Figure VI). After some years the glands become nodular and vary greatly from part to part (Stuart, 1955; Taylor, 1956). In the parenchymatous goitres of the Alps and Himalayas, the swelling is due to increased height and number of cells, so that the gland resembles that seen in mild Graves's disease (McCarrison, 1937). All variations between this and colloid goitre are found.

Pathogenesis.

The steps in the transition from a normal thyroid to one with colloid goitre are not quite clear. The usually accepted explanation is that of Marine (1935). He considered that the amount of thyroxine secretion fell, because the gland was hindered by lack of iodine and sometimes also by the effect of ingested goitrogens. That, he thought, led to increased secretion of thyrotropin and so to hypertrophy and even hyperplasia of the thyroid cells, with the formation of a parenchymatous goitre. Subsequently, in most cases, the secretion of thyroxine became sufficient because of (*a*) increased intake of iodine, (*b*) decreased intake of goitrogens, (*c*) more efficiency in the altered gland, (*d*) decreased requirement of hormone, or because of a combination of these factors. When this happened, Marine stated that colloid collected in the vesicles, and the cells became flattened and smaller. He called this an involuted thyroid, and held that it represented the closest approach to normal which a thyroid that had been actively hyperplastic could again assume. A thyroid gland enlarged by endemic goitre was known to enlarge further with puberty, with pregnancy and at other times. When those temporary stimuli had ceased, the gland shrank, but remained larger than before. Marine attributed the swelling to a change-over to a parenchymatous goitre. He attributed the subsequent shrinking to reversion to a colloid type (Marine and Lenhart, 1909).

There are a number of arguments in favour of Marine's hypothesis.

1. The study of the normal thyroid has shown that when prolonged activity is followed by respite, the vesicles of the gland become distended by colloid and the lining cells flattened. From this it has been argued that the changes in diffuse colloid goitre indicate that there has been a previous stage of excessive hyperplasia, and that the stimulus which caused this has now ceased, the result being hyperinvolution.

2. Animal experiments have shown that diets deficient in iodine may lead to hyperplasia and enlargement of the thyroid gland. This is more rapidly and readily achieved if the food also includes goitrogenous substances, such as cabbage and lucerne (Marine, 1935). Marine and Lenhart (1909) described colloid goitre in dogs in a goitrous district. They found all transitions between active hyperplasia and colloid goitre. They also produced a change from active hyperplasia to a colloid goitre by giving iodine.

3. The parenchymatous goitres of the inhabitants of some mountainous areas often pass to a colloid stage (McCarrison, 1937). In these patients the gland passes through the succession of changes which Marine postulated.

4. Another observation is that of groups of tall cells in the lining of some alveoli in a colloid goitre illustrated in

Figure VI. These are taken by some to represent the remains of a previous hyperplastic stage (Means, 1948).

There are also some strong arguments against the validity of Marine's hypothesis.

1. Animal experiments can never completely prove the course of a disease in humans. It is known, for instance, that in dogs, which have been used a great deal in these experiments, typical nodular goitre does not occur (Mellanby, 1934). This is good evidence that the thyroid glands of dogs are not entirely analogous to those of men. Mellanby (1934) gave a group of pregnant bitches a diet deficient in iodine and containing cabbage. The puppies were subsequently fed on the same diet. When their thyroid glands had become enlarged by hyperplasia he gave them iodides. Animals given a low dosage of iodides developed colloid goitre. Those given a larger dose showed a reversion of their glands to a normal state.

2. After fifty years, Marine's theory has not been substantiated by the collection of glands at the stages through which he postulated that they passed in the development of diffuse colloid goitre. In Australia and in America, diffuse parenchymatous goitre is not seen in thyroid glands removed for endemic goitre (Stuart, 1955; Bundock, 1956; Hellwig, 1932). The few small groups of tall cells which are often seen form an insignificant proportion of the gland.

3. Hellwig (1932) in America reported that the enlarged glands in pregnant women and at puberty have the structure of a colloid and not a parenchymatous goitre as Marine postulated.

From all this it must be concluded that Marine's hypothesis is not true for the development of diffuse colloid goitre, at least in Australia and America. In these countries, and in all but the most severely affected endemic areas elsewhere, the thyroid gland passes to a colloid goitre simply by accumulating excessive amounts of colloid. At the same time the cells become flattened. There is no proof, then, that there is a preliminary stage of parenchymatous goitre in the development of colloid goitre in these countries. In the severely affected parts of the European Alps and in the Himalayas the story appears to be different.

The development of diffuse colloid goitre in sporadic cases has not been studied in the present work, but it is assumed to be the same as in the endemic cases.

The Nature of Colloid Goitre.

The nature of the changes in colloid goitre has been obscured by two words which have been used loosely. "Hyperplasia" should mean increase in the number of parenchymal cells; it has often been assumed to mean increased activity of the gland in producing hormone or in endeavouring to do so. "Involution" has been used to mean both lowering in the height of the cells (and so in the size of the gland) and also reduction of the activity of the gland. However, there is good evidence that the size of the thyroid cell is not always proportional to its activity. For instance, patients may exhibit hyperthyroidism without enlargement of the gland (Greer, 1955). Halmi (1954) found changes in activity without changes in cell size in rats.

When Marine stated that a colloid goitre was a thyroid gland in involution, he meant that the cells were low and assumed that this indicated low activity. Anderson and Winship (1953) described such a gland as exhausted.

However, the nature of the altered gland is not so simple. It is a more efficient iodine trap than a normal gland, as was shown by Taylor (1956) and by Stanbury (1955) in tests on the uptake of radioactive iodine. That is, if the patient is given a dose of iodide by mouth, the colloid goitre will remove it more rapidly from the blood than would a normal thyroid. Stanbury (1955) also found that the disappearance rate of the radioactive iodine from the gland was unusually slow, and he attributed this to rapid turnover and reutilization.

The gland is, or has been, working under the difficulties of low iodine intake and of the effects of goitrogens. The increased ability to trap iodine and to use it over and over again helps it to secrete more thyroxine than would a normal gland under such conditions. Thus signs and symptoms of thyroxine lack are rare in patients with colloid goitre, and normal levels of protein-bound iodine are usually found in the serum (Taylor, 1956). We must, in the light of this evidence, regard the changes of colloid goitre as more complex than just exhaustion, involution or atrophy.

In the past we have considered the gland to be controlled almost entirely by thyrotropin, an excess of which in the blood led to increased secretion of thyroid hormone with an increase in the size and number of cells if this condition was maintained for long. Lack of thyrotropin meant less secretion and smaller cells.

However, this explanation cannot be extended to explain the low cells of colloid goitre, which are very efficient in the directions described and are by no means inactive. One possible explanation is that thyrotropin is composed of two factors (Greer, 1955). One of these is said to be the growth factor, which stimulates the growth of thyroid cells. The secretion of this factor is dependent on the integrity of a specific hypothalamic area. It is postulated that some substance passes from the hypothalamus to the pituitary by a portal system of blood vessels (Harris, 1951). The other thyrotropic factor is a metabolic factor, which stimulates metabolism of iodine by the thyroid and appears to be independent of the hypothalamus. Both factors, at least in part, appear to be regulated by the concentration of circulating thyroid hormone. Greer suggests that the occurrence of goitre without evidence of increased formation of hormones may be due to increased rate of secretion of the growth factor alone. This would explain the findings in colloid goitre. This increased secretion of growth factor might be due to hyperfunction of either the pituitary gland or the hypothalamus. Symptoms of hyperthyroidism with an apparently normal gland may be caused by an increased rate of secretion of the metabolic factor with no increase in the growth factor. This hypothesis is not yet proved.

Other explanations of the changes in colloid goitre include a direct effect of iodine lack on the thyroid gland without involvement of the pituitary or hypothalamus (Astwood and Solomon, 1955).

Summary.

From the evidence it is concluded that, in Australia at least, no hyperplastic stage occurs in the formation of a colloid goitre. A colloid goitre is a thyroid gland poised to grasp and use efficiently a high proportion of any iodine ingested.

Attention is drawn to the theory of the pathogenesis of colloid goitre, which postulates an imbalance between two factors that go to make up the thyrotropic hormone.

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SALIENT POINTS IN THE DIAGNOSIS AND TREATMENT OF TRAUMATIC LESIONS OF THE KNEE JOINT.[1]

By LENNOX TEECE,
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WE are met here tonight for one common purpose—to do honour to the memory of Arthur Meehan. To many he is known chiefly by reputation as the founder of orthopædic surgery in Queensland and as a surgeon of outstanding merit and judgement tutored by years of experience. To some of us there were the closer, more personal bonds of friendship forged over many years of association in a common pursuit. From the bottom of my heart I thank you for the privilege of giving this inaugural Meehan Memorial Lecture.

I should like to think that I have been so invited, not because I am the senior practising orthopædic surgeon in Australia, but because I was one of Meehan's oldest friends. Many a readier tongue than mine could have been found to deliver this lecture. Though I lack the eloquence to translate into vivid language the thoughts and the memories that crowd the brain when we think of the friend and surgeon we have lost, I yield to no one in sincerity and I voice the feelings of us all when I say that we have lost a good friend and Australia has lost a surgeon of note, a man of great merit and stature, whose place will not easily be filled.

Tonight I would ask your attention while I discuss some of the salient features in the diagnosis and treatment of traumatic lesions of the knee joint. Undoubtedly the commonest condition encountered in the knee is a lesion of one or other of the semilunar cartilages or menisci. We have at last passed beyond the age when every patient with an injured knee joint was certain to be told by someone that he had a torn cartilage. It has been replaced by the present age when every patient with a painful back is told he has a prolapsed disk.

In the past I have frequently been amazed at the manner in which, in some quarters, the diagnosis of a displacement of the internal semilunar cartilage is made without any adequate evidence. On the other hand, it is equally an error to refuse to diagnose this condition without a history of definite locking of the joint. The classical description of the condition, as given in the standard textbooks, always attaches much weight to the history of locking, but is apt to convey the impression that this must happen in every case of cartilage injury. This, of course, is by no means the case.

My observations are based on a study of two thousand patients on whom I have operated with the diagnosis of injury to a cartilage. In all large series of cases there is a definite, though varying, proportion of diagnostic errors. Mine has been 4%. The internal semilunar cartilage is injured approximately 12 times as often as the external one. Both cartilages may be injured in the same accident, and at times it is difficult to determine which cartilage has been damaged. It is wrong to assume that the primary displacement can be the result only of severe violence. The appropriate movement to produce displacement of the internal semilunar cartilage is abduction of the leg on the thigh combined with external rotation of the tibia on the femur. This movement can produce a typical tear of the cartilage when carried out with quite a small degree of force.

I encountered one man who climbed up a fixed iron ladder out of a lift well. Just as he was emerging therefrom and putting his left knee onto the floor, his right foot slid outwards along the top rung of the greasy ladder, this right leg being abducted on the thigh. He felt a sharp pain on the inner side of the right knee joint, which locked in typical fashion, and operation subsequently revealed a typical, bucket-handle tear of the internal semilunar cartilage. In this instance the degree of violence was quite trivial.

Tears and displacements of the cartilages may occur without any history of injury and without evoking any reaction such as effusion of fluid. I have operated on three patients in whom the condition occurred in the internal semilunar cartilages of both knee joints without any history of injury whatsoever.

One of them was a man whose work necessitated constant kneeling whilst nailing down floor coverings. He gave a history that on one day, as he was rising to his feet, one knee locked. He released it himself after a little manipulation. No effusion followed, and he continued to work without difficulty. This locking was subsequently repeated frequently, and sometimes occurred three or four times a day. After the lapse of a few months, identical symptoms made their appearance in the opposite knee, and at operation I found that both internal semilunar cartilages were ragged and torn.

In addition to these bilateral cases I have encountered many others in which one knee only was affected, the original locking having occurred spontaneously, and I have been struck by the fact that in many of these men the initial locking occurred when they were rising from the squatting position. Locking does not necessarily occur as a primary symptom. Often the knee sustains a minor twist. It appears to be slightly sprained and is a little painful and perhaps faintly swollen; but the worker carries on and it is not till some time later that actual locking occurs.

I remember a timber-getter, camped on the job in the bush, who gave his knee a minor twist, which he disregarded. He went on working, though the joint was slightly painful. Early the next morning, in the entire absence of any sanitary conveniences, he retreated deeper into the bush and assumed the squatting posture. On his attempting to rise, the knee locked, the locking being accompanied by acute pain, and he fell over backwards with unfortunate results.

I have observed a large number of patients who sustain injury to one internal semilunar cartilage which is removed surgically, and who, after an interval of months or years, sustain a similar injury to the cartilage of the opposite knee. In most of these people both injuries have been of a trivial nature, and I hazard the suggestion that some cartilages are congenitally only loosely anchored in position.

Beware of the word "locking" when used by the patient. I never use it in his presence. A history of locking should be subjected to very close cross-examination. Every patient is familiar with the word. He often volunteers the statement that his joint has locked when it has done nothing of the kind. He has injured the knee; his joint is swollen and painful and therefore naturally most comfortable in the slightly flexed position. He has no intention of moving it into full extension, thereby giving himself pain. He says that his joint locked and he could not extend it; but he has confused "could not" with "would not". Frequently also the patient will apply the term "locked" to a joint in which flexion is limited by pain. Accordingly, one must

[1] The Meehan Memorial Lecture, delivered under the auspices of the University of Queensland Post-Graduate Medical Education Committee on November 30, 1956, at Brisbane.

always ascertain exactly what the patient means when he describes the joint as having been locked.

A history of definite unlocking with the immediate restoration of full extension of the joint and very considerable relief of pain is much more valuable from the diagnostic standpoint, for this is not a symptom about which the patient can be mistaken. The ease or difficulty with which a knee can be unlocked varies greatly in different patients and in the same knee at different times. An unskilled layman will often succeed in unlocking a joint by making traction on the leg combined with extension of the knee joint. The experienced patient, who has suffered from repeated displacements, usually learns to unlock the joint for himself, and does so with less difficulty and discomfort than when he trusts the task to his medical attendant, chiefly because, having confidence in his own manipulation, he relaxes his muscles completely at the onset. The usual self-manipulation consists of resting the balls of the toes lightly on the ground and combining circumduction with gradual extension. However, many knees can be unlocked only when complete relaxation is secured under an anæsthetic, and even then this procedure may offer much difficulty.

The manœuvre usually advised is to flex the knee fully, abduct and externally rotate the leg on the thigh and then sharply and simultaneously internally rotate and extend the leg. Success will be more readily obtained if after the knee has been flexed the leg is circumducted before the remainder of this manipulation is carried out. Long-continued persistent locking of the joint, or in other words persistent displacement of the central part of the internal semilunar cartilage towards the intercondylar notch, influences the prognosis badly. It sets up chronic traumatic arthritis with persistent effusion, synovial thickening and limitation of movement, which may not subside after removal of the cartilage. When attempts are made by manipulation to reduce the displacement and to undo the locking of the joint, a pseudo-reduction is often effected, the central part of the cartilage moving still further towards the centre of the joint, so that it comes to lie in the intercondylar notch, but no longer blocks extension. There is no possible method of knowing whether a true reduction or a pseudo-reduction has been effected. This point is of course of great importance when the advisability or otherwise of conservative treatment is being considered.

Do not accept the patient's statement that a joint has unlocked, or in other words that extension has been restored. He is often mistaken. When he is examined in the acute stage, extension of the knee may be limited by as much as 40°. The major part of the limitation is due to muscle spasm of the hamstrings. As the acute symptoms subside, extension is gradually partially restored as the muscle spasm relaxes. The actual mechanical block to full extension caused by a displaced cartilage does not exceed more than 5° or at the most 10°. When the lack of extension has been thus reduced, the patient often believes that full extension has been restored, whereas in reality the joint is still locked. Indeed, in many patients examined in the quiescent stage after the effusion has subsided and the greater part of the pain departed, though the joint is still locked the degree of limitation of extension may be only minute, and the range of movement of the affected knee requires close comparison with its fellow. This minute lack of extension is accompanied by a characteristic feeling of an elastic resistance to the performance of this final fraction of extension.

In the present state of modern surgery I believe that when the diagnosis is unequivocal the cartilage should be removed in every case, even in the presence of pronounced preexisting osteoarthritis. However, there will be some patients who refuse or are otherwise unsuitable for operation, and for such people three weeks rest in bed is ordered after a manipulative reduction combined with quadriceps exercises from the outset.

The variety of possible tears of a cartilage are legion, but the two main types are the bucket-handle tear and the posterior horn tear. I describe them respectively as the cartilage of symptoms and the cartilage of physical signs. The bucket-handle tear produces a typical history of locking followed by unlocking, sudden or gradual, and when the patient is examined in the quiescent stage in the interval between attacks, there may be an entire absence of physical signs. On clinical examination the joint may appear normal. However, in the presence of a clear-cut history and after one has satisfied oneself that locking of the joint really did occur, there is no hesitation in making a diagnosis and in advising operation. The bucket-handle tear is the cartilage of symptoms. However, locking or limitation of extension of a knee joint may be due to many causes other than a displaced cartilage. The degenerated knee joint of the elderly labouring man often lacks 10° of extension. This will be true of both his knees. The knee whose internal lateral ligament has been sprained is the most difficult of all to distinguish and is responsible for nearly all the errors in diagnosis.

In many such cases extension is limited by a few degrees; but there are usually localized thickening at the medial joint line over the internal lateral ligament and tenderness over the adductor tubercle. In a cartilage lesion the tenderness is more anterior. These are minor differences in signs, so it is small wonder that some mistakes in diagnosis creep in. The patient on whom operation is performed and from whose knee a normal cartilage is removed, and who yet makes a complete recovery, has all along been suffering from a damaged internal lateral ligament. Make no mistake, once having put your hand to the plough you cannot turn back. Having opened the joint with a diagnosis of a cartilage lesion, you must proceed to remove the cartilage unless some other obvious condition is encountered. You cannot describe a cartilage as normal until you have almost finished removing it.

In many cases of posterior horn tears, the cartilage at first appears normal when the joint is opened, and it is not till the anterior horn has been divided and the dissection round the periphery half completed that the posterior horn tear becomes evident. Other causes of limited extension are fractures of the spine of the tibia and the presence of loose bodies in the knee joint.

The necessity for an X-ray examination in every case is obvious and should establish the presence or absence of either of these conditions, always provided that the X-ray picture has been taken by a competent radiologist and not by a tiro who is engaged in purchasing his first machine on easy time payment. The fracture of the spine of the tibia avulsed by the pull of the anterior cruciate ligament and blocking extension occasionally occurs as the result of surprisingly little violence.

In young people the commonest source of loose bodies is *osteochondritis dissecans*, which always affects the same area of the articular surface of the internal femoral condyle, though the size of the resultant loose body or bodies varies greatly. This area, by the way, is the area in which the first sign of degeneration of the articular cartilage makes its appearance in the incipient osteoarthritic knee. Of course, we have no adequate reason to regard osteochondritis as a traumatic lesion; yet patients so readily produce a history of injury, often imagined, that even in this disease we are usually told of some trauma.

There is the occasional loose body which is purely cartilaginous and does not show in the X-ray film. I have just recently removed a large loose body of this type from a young girl whose internal semilunar cartilage had already been removed by one of my colleagues. I take no credit for the diagnosis. It was made by the patient herself before I saw her. She felt it with her own fingers.

Obviously I make no mention of the limitation of extension caused by the chronic diseases such as tuberculosis or rheumatoid arthritis. One small point before I leave the sign of limitation of extension: limitation of extension in the presence of free, unrestricted flexion is almost diagnostic of a displaced cartilage, but is, of course, found only after the acute symptoms have subsided.

Having thus dealt with the bucket-handle tear—that is, the cartilage of symptoms—I shall pass on to the posterior horn tear, the cartilage of physical signs. In the presence of a posterior horn tear a cartilage becomes unduly mobile, moving forwards and backwards on the appropriate move-

ments of the joint. In the acute stages the symptoms of all cartilage injuries are largely the same—effusion of fluid, pain on the medial side and limitation of all movements due to pain. The patient may, at the time of injury, feel that something has momentarily become displaced within the joint, but does not experience true locking. Subsequently the cartilage is apt to become habitually mobile, and the patient is conscious of this mobility. Different patients describe it in varying through similar terms. Some say that they are frequently conscious of a momentary catch on the inner side of the knee. Others say that they constantly feel a click in this situation; but do not be misled by the common and innocuous patellar click. Others again describe the sensation as of two knuckles rubbing past one another. The degree of mobility varies greatly. In extreme cases it can be palpated every time the patient flexes and extends the knee as he lies on the examination couch; in other cases it is here today and gone tomorrow. I have often suspected a patient of possessing a mobile cartilage, but have not been able to feel the mobility. I have told him to come back on another day, and have subsequently felt the cartilage mobility with the greatest of ease.

The best known, but not the best, method of palpating this mobility is the well known manœuvre of McMurray. This consists of rotating the acutely flexed tibia on the femur whilst the patient is recumbent and also adducting and abducting the leg on the thigh. If this fails to elicit any mobility the leg is slowly extended at the knee joint, whilst at the same time it is abducted and externally rotated. The weakness of McMurray's test is that it cannot be employed if the knee is too painful and swollen to permit of full flexion, or if the patient is too apprehensive to allow this movement.

The test I have devised can be used under all circumstances, provided that about 40° of flexion is obtainable. The patient is instructed to advance the foot of the affected leg, standing in the position which would have been reached if he had just lunged forward on to that foot. The knee should be slightly flexed and there should be sufficient weight on the foot to prevent the heel from leaving the ground when the knee is manipulated by the examiner; but there should be insufficient weight to prevent free manipulation of the joint. The other knee is in full extension and the limb is externally rotated. The surgeon sits on a low chair alongside the patient and embraces the knee joint with his two hands, the tips of the fingers being placed on the joint line on the inner side. He then circumducts the knee in both a clockwise and an anticlockwise direction, and a sensation is felt beneath the examining fingers as of something suddenly and momentarily jumping out of position. The patient can also quite well appreciate the moment at which this occurs.

This cartilage click is not to be confused with the constant grating one feels on circumducting the osteoarthritic knee. It may be necessary to manipulate the knee for a minute or two before the sign can be elicited, and indeed, as was mentioned before, it comes and goes with unpredictable inconstancy. When you feel the sudden movement of the cartilage beneath your fingers the patient will usually tell you that that is the catch or click of which he has been speaking. I have found that the mobility of a torn cartilage is much more easily and more constantly detected by this method than by McMurray's test.

Bucket-handle tears and posterior horn tears occur with about equal frequency. There are numerous other types of tears, but all these others are rarities. An internal semilunar cartilage click is practically never audible. To explode another fallacy, there is no such thing as a circumferential detachment of an internal cartilage. Every so-called circumferential detachment is really a bucket-handle tear, the torn portion comprising nearly the full width of the cartilage, and if one looks closely at operation one will see an extremely narrow rim still attached to the deep surface of the capsule. The cartilage has been torn, not torn away.

Thus in the cartilage of symptoms we have the history of locking, and sometimes, and better still, the history of unlocking. In the cartilage of physical signs we have the palpable mobility of the cartilage. If both these features are absent it is rarely justifiable to operate. In the acute stage immediately following a cartilage injury, the making of a diagnosis is often for a time impossible. The joint is painful and swollen and lies in slight flexion, and all movements are resisted. It is impossible to determine whether the joint is locked or to carry out tests for undue mobility without examining the patient under an anæsthetic. It is better to delay the examination for a few days till the joint is more manageable. Abnormal mobility, either in a lateral or in an antero-posterior direction, due to damage to the lateral or cruciate ligaments, gives a feeling of insecurity to the knee, and this insecurity is often described by the patient as a sensation of something moving in the knee joint. This ligamentous damage is not likely to be missed when the physical examination is conducted. In this enlightened age no one is likely to mistake the sesamoid bone or the fabella in the outer head of the gastrocnemius for a loose body in the knee joint.

I had a patient sent to me from a country town, accompanied by his X-ray films, with a diagnosis of loose body in the joint. The so-called lateral view had been taken in a very oblique plane, and in it the sesamoid bone appeared to lie well within the joint cavity. Its size and shape gave it away; but nevertheless a candidate for the degree of master of surgery fell into this obvious trap when presented with the X-ray film. Incidentally, the patient had a bucket-handle tear of the cartilage.

Rarely it may be months before an exact diagnosis can be established. One suggestive sign in a young, otherwise healthy patient is the occurrence of repeated effusions into the knee joint for months after apparent temporary recovery from what has been regarded as a simple sprain of the joint. Even then, if one waits long enough, the day will come when either the joint will lock or palpable mobility of the cartilage will become evident.

The technique of operation in the hands of different surgeons varies only in minor detail. Nobody now, I trust, ever uses a Bard-Parker knife within the joint, though it was, until recently at any rate, employed in one Sydney hospital. The danger of breaking the blade, or equally of breaking the blade of a tenotome, if one is foolish enough to employ it to divide the posterior horn, is not a risk to be taken. In the closing of the incision in the synovial layer the sutures must be carefully placed in its distal part. They not only shut off the joint cavity; they are hæmostatic. All the vessels are in this lower part of the incision and if they are not properly secured the joint will fill with blood on removal of the tourniquet. To make a second, a posterior, incision to remove the entire posterior part of the cartilage is superfluous and unnecessary. I have known recurrent symptoms after operation to be attributed to the presence of the remaining posterior end of the cartilage; but in the few patients I have examined whose joints had been reopened to remove the posterior end of the cartilage, the symptoms, which were vague, were not ameliorated.

I always apply a back splint with the knee in full extension for five days after operation. It is not necessary, and I secretly feel that the real reason I use it is that one must see and remove the tourniquet when applying the back splint. I have always suffered from a phobia with regard to the possibility that a tourniquet may be left *in situ*, and the few such cases that have been reported constitute a major tragedy. An unpleasant complication I have seen on five occasions is temporary paralysis of the external popliteal nerve due to a compression bandage applied too tightly, or more accurately, applied badly. This comes on a few days after operation. The careless applicator has placed ample cotton wool on the front of the joint, with but scanty padding over the head and neck of the fibula and the hamstring tendons. This paralysis always recovers, but may take four or five months to do so. An occasional complication is a synovial leak, usually occurring at the end of the first week, after active movements of the knee have been commenced. One is met by the sister with the breathless announcement that the wound is discharging. Some sisters are designed by nature to be the bearers of bad tidings.

On removal of the dressings the edges of the wound are red and shiny and a little synovial fluid may be leaking out, or it may not actually leak out till a day or two later. However, there is no appreciable pain, no rise of temperature, and most importantly, there is no pain when the joint is placed in full extension. If a knee joint can be placed in full extension without intense pain being excited there is no septic condition within that joint. Synovial fluid appears to be very irritating to skin.

Lesions of the external semilunar cartilage produce symptoms which closely parallel those of the internal cartilage, if allowance is made for the difference in location. In my experience they have occurred in the ratio of one to 12. Here again one has the two main types—the cartilage of symptoms and the cartilage of physical signs. One meets with a bucket-handle tear which causes locking of the joint, and the posterior horn tear resulting in palpable mobility. In this case the mobility is much more dramatic than in the case of the internal cartilage.

On manipulation of the knee the head of the tibia seems to jump forward on the femur, and this is often accompanied by a loud click. In the cartilage of symptoms seen in the quiescent stage it is occasionally impossible to determine which cartilage is at fault. I have on five occasions removed a normal external semilunar cartilage when the symptoms were all located to the outer side of the joint, and have later removed from the same joint an internal cartilage showing a bucket-handle tear. I can offer no explanation why, in these cases, the symptoms were referred to the side of the joint opposite to the situation of the lesion.

The congenitally discoid lateral cartilage met with, usually in children, sometimes in adults, does not produce symptoms unless or until it is injured. The fact that the vast majority of cases are met with in children proves that such a cartilage is vulnerable to slight degrees of injury, and the patient seldom reaches adult life without its being damaged. When one is presented with the typical history of the loud clicking knee, make the child squat tailor-fashion on the floor with crossed legs. The loud click will then frequently be elicited as he commences to rise to his feet again.

Cysts of the internal cartilage are rare, those of the external cartilage are relatively common. The symptoms are vague, consisting of aching pain at the lateral aspect of the joint. These cysts are always unmistakable, the visible portion being constant in size and location. It is not, as is so often stated, in the middle of the lateral surface of the joint line beneath the lateral ligament. It is anterior to this at the joint line just above and in front of the head of the fibula, and the visible and palpable portion is, in size, the section of a small marble. It is phenomenally rare to find it any larger. The greater part of the multilocular cyst is, of course, hidden from view within the joint, and obviously the whole cartilage must be removed.

I should like to draw attention to one or two points in connexion with fractures of the patella. These fractures fall naturally into the following two groups—the transverse fracture from muscular violence, which divides the bone into two more or less equal halves, the fracture line being transverse, and the comminuted fracture, in which the bone is broken into multiple fragments. All are agreed that the simple transverse fracture calls for suture of the bone, the use of catgut, kangaroo tendon or wire depending on individual taste; but do not imagine that accurate apposition of the fragments can be secured without actually drilling the bone and passing the sutures through it. The old-fashioned U-shaped incision has been abandoned, not only because it is apt to result in sloughing of the skin at the distal end of the flap, but also because it does not fully expose the transverse rent in the aponeurosis and the capsule, and because suture of the quadriceps expansion is just as important as suture of the bone.

In the case of the comminuted fracture of the whole bone one must carry out a complete excision of the patella without suffering from the delusion that the extensor power will not be weakened thereby. When there is a fracture, sometimes comminuted, of either the upper or the lower pole of the patella and the remaining two-thirds of the bone are intact, the temptation is to remove merely the fractured fragments, and most authorities recommend this course. They are wrong. If the fragments are such that accurate suture and restoration of the smooth contour of the articular surface of the patella are not possible, the whole bone should be excised. Removal of only the upper or lower pole of the bone results in poor restoration of movement and the development of severe patello-femoral arthritis.

I should like to say something about a rare and serious injury that is seldom mentioned, and if it is mentioned at all is described inaccurately in the text-books. I refer to avulsion of all or part of the head of the fibula. The injury is produced by violent adduction of the leg on the thigh, and the head of the fibula is torn off by traction of the external lateral ligament and displaced proximally. It is always complicated by paralysis of the external popliteal nerve, which may be actually torn in two. The immediate treatment is obvious—open reduction and internal suture of the head of the fibula back into its normal position. The important feature is that whether the external popliteal nerve is torn or appears normal to the naked eye, it never recovers, and the paralysis is permanent. I have encountered four such cases all resulting in permanent paralysis. When one considers that, after all, suture of a divided external popliteal nerve carries with it almost as good a prognosis as suture of a radial nerve, one is completely at a loss to explain the hopeless prognosis when it is injured in association with this fracture.

Fractures of the tibial plateau are very common injuries, and in this motor-ridden age a blow from the bumper bar of a car is the common causal factor. Nearly always the damage is to the lateral tibial condyle, and the fragment is depressed and the articular surface broadened from side to side. The fracture as a fracture matters but little. By all means attempt to replace the fragment as best you may. Traction combined with side-to-side compression of the bone in a clamp will often improve the position. Whether or not accurate reduction is secured, union will take place and, except in the elderly, excellent movement will be regained, and it is rare for any disabling degree of traumatic arthritis to supervene. The crucial point in this type of fracture is: is there or is there not associated gross damage to the lateral or cruciate ligaments? This point can be determined only by examination under an anæsthetic. If such is the case, there is a danger of permanent instability of the knee joint. In this as in many other knee joint injuries our sheet anchor is the quadriceps muscle. During the eight weeks' necessary immobilization in plaster and afterwards, active, frequently repeated quadriceps exercises must be insisted upon, including weight-lifting exercises. Open reduction of these fractures is rarely called for and usually effects nothing. The small fragments will only be further broken up by the insertion of a screw, and when they are replaced will decline to stay in position.

Avulsion of the tibial spine from traction through the anterior cruciate ligament is not an uncommon injury. This injury is not, as has often been alleged, confined to the young. It may occur at any age. The displaced fragment limits extension by 5° or 10°. If, under an anæsthetic, the knee is forcibly fully extended, the fragment will regain its original position, and this reduction can be effected when one or two weeks have elapsed since the time of injury. Watson-Jones advises that in difficult cases the joint be opened and the fragment be pressed into the bed from whence it came, and states that it will be stable without any fixation. Only a super-optimist would agree with him, especially if the fracture is of more than two weeks' standing.

Speaking to an audience of practising surgeons, I have not attempted to deal exhaustively with any of these traumatic lesions of the knee joint. Rather I have endeavoured to focus attention on certain practical points of diagnosis and treatment. Over the years we have all learnt the hard way, and if these remarks, learnt from the experience of 2000 cases and from my own mistakes, prove of any value to you who have the handling of these cases, my efforts will have been well spent.

It is indeed fitting that his Queensland friends decided to found a memorial lecture in honour of Arthur Meehan, or "Paddy" as all his friends knew him. When the history of orthopædic surgery in Australia comes to be written his name will be foremost in the records. Graduating from the University of Sydney in 1914, he served as a resident medical officer at Sydney Hospital and then went to the First World War in 1916. He lost a leg at Passchendaele in 1917, but employed his convalescence to good purpose, working in Liverpool, the home of British orthopædic surgery, and he also gained his Fellowship of the Royal College of Surgeons of Edinburgh. After working for a while at Rosemount Military Hospital he commenced practice in Wickham Terrace in 1920, and there he spent the rest of his professional life. He was the pioneer of orthopædic surgery in Queensland, founding the orthopædic departments at the Brisbane General Hospital, the Brisbane Children's Hospital and the Mater Misericordiæ Hospital. He was one of the first three Australian-born orthopædic surgeons, and a foundation member of the Australian Orthopædic Association and for some years its president. He also rendered notable service as chairman of the University of Queensland Post-graduate Medical Education Committee. When some few of us were engaged in a struggle for the recognition of orthopædic surgery in Australia, Meehan was in the forefront of the battle. He disarmed opposition till he lived to see orthopædics accorded more universal recognition in Queensland than in the other States. He lit the torch of orthopædic surgery in Queensland and fanned its early flickering to a bright flame of achievement.

We need no structure of stone or brick to perpetuate his memory: *Si monumentum quaeres circumspice.* His monument is the school of young, trained orthopædic men—young men of ability—now carrying on the work he started. To them he was always a beacon of inspiration, guiding them along the path of sound surgery and avoiding the showy and the ephemeral. What a man does with his own hands and his own brain during his short stay on earth is but a little thing. The inevitable hand of time reaches out and takes him, his associates and his patients. His claim to remembrance lies in what he builds for future generations, and Meehan built around himself a school of young men with a high standard of orthopædic knowledge that will not be easily surpassed in any other centre.

As a man and a friend he was fair-minded and generous, tolerant in thought, but always a fighter for his convictions. His tragic death at the hands of one mentally deranged occurred when he was still at the zenith of his powers. It has left a gap that will not easily be filled in the ranks of orthopædic surgeons in Australia, and still more in the hearts of his friends. His memory still lives with us. I cannot conclude better than by using the immortal words that Mark Antony spoke of Brutus:

> His life was gentle; and the elements
> So mix'd in him that Nature might stand up
> And say to all the world,
> *This was a man!*

COOPERATION IN A GENERAL HOSPITAL COMMUNITY.

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GRANTED that if common goals are to be reached cooperation is essential in any community, and granted further that in a general hospital community the life of not only one patient, but groups of patients, may depend upon the ability of the personnel to cooperate, what, then, are the factors that make for problems of cooperation in any community? In a hospital community? Specifically, how may the almoner working in a general hospital setting help to promote good interpersonal relationships in the hospital community?

During the years different definitions have been attempted in an effort to delineate what is meant by the term "community". In the more recent text-books of sociology one finds increasing emphasis upon definitions that analyse the concept of community in terms of structure and function. This is a valuable approach when one wishes to study such problems as housing, delinquency, mental illness and many others often referred to as community problems. An awareness of structure and function provides a framework for analysis which, according to some sociologists, includes the following dimensions (Odum, 1951).

The Framework of Structure and Function.

Geographical Base.

Every community has its geographical base—its climate, amount of sunshine, potential fertility of soil and other physical attributes. It also has its geographical boundaries. They may or may not be definite, but they do exist. A general hospital, too, has its physical base, its climate and other elements that give it geographical dimensions. It also has its boundaries. The immediate limits are the walls and grounds of the hospital buildings. The larger boundaries mark the geographical area served by the hospital. For purposes of this paper we shall consider only the geographical boundaries as limited by the walls and grounds of the hospital buildings.

Population Vitality, Composition and Trends.

In a study of a community the sociologist always concerns himself with the people in it. Among the questions he wants answered are such as the following: How many people are there? Who are they? What nationality and age groups are represented, and in what numbers? What is the proportion of men to women, single to married? What is the rate of mobility of the population and in what direction is it moving? Are more persons coming in or going out? How are the newcomers adjusting themselves to the rest of the population? There are a host of other questions. These same inquiries are probably included in most studies of general hospital communities.

Ecological Patterning.

In a community people arrange themselves in relation to streets, hills, streams, beaches, nearness to business houses, schools, churches and parks, and in relation to other factors. A population map shows these patterns in graphic form. Patients and staffs in hospitals form similar patterns. They are grouped together in wards, departments, offices, waiting rooms, floors, laundries, dining rooms, kitchens *et cetera.* These patterns have great significance. Take, for example, the decision of a general hospital to admit a group of patients with an infectious or contagious disease, such as tuberculosis or poliomyelitis. The ecological patterning becomes very important. It may mean the difference between spreading infections or not spreading them. Similarly, the ecological patterning of patients with acute illness and those with chronic diseases may make quite a difference in how each group responds to treatment.

Institutional Structure.

The heading "institutional structure" includes formally organized groups as well as groups of people informally bound together because of common interest. There are the Rotary Clubs, the Association of Almoners, the branches of the Australian Red Cross Society, and many other formally organized associations. There are also groups that may not even consider themselves to be groups. They may be cliques, gangs, informal associations, without charter, constitution or officers. They are people held together because of common interests. They may be as powerful as any organized groups, and they may have to be reckoned with in a given situation quite as consciously as any organization with by-laws and a duly elected president and secretary.

[1] Read at the annual meeting of the Australian Association of Almoners on May 19, 1956.

A hospital, too, has this kind of institutional structure. The hospital has departments, divisions, committees and other formalized associations. It also has its cliques, its aggregation of individuals who, because of special interests or personality patterns, or for many other reasons are banded together. In a hospital as well as in any other community they may wield potent influences, both constructive and destructive.

Social Stratification and Vertical Mobility.

In every culture, primitive or otherwise, there seems to be some stratification of the people in the community. Even in our countries, said to be most democratic, the people in them do not have the same status. Some have high status, some less high, and some low—sometimes very low. The stratification may be related closely to the role of the individual. Perhaps the tiller of soil in a primitive society has more status than the hunter or fighter. Then again the situation may be reversed. The mechanic may have higher status than the clerk, the plumber may rank higher than the college professor. Sometimes the status depends upon financial income, real or assumed. It may make a difference in his status if a man earns £5000 rather than £500 a year, or if his wife wears a fur cape rather than one made of cloth.

In a hospital, status definitely plays a role, both among staff and among patients. The patient of high status may or may not demand more and may or may not get more than the patient of low status. The status of staff is often dependent upon role—the role of doctor, nurse, kitchen helper *et cetera*. In a community in which group relationships have a democratic base there is often opportunity to move from one status to a higher status. The sociologist calls this vertical mobility. The almoner of 1956 has a higher status in an Australian hospital than had the almoner of twenty years ago. Australian almoners have experienced vertical mobility, as have other members of a general hospital staff.

Power Structure.

A community has a system of both delegated and undelegated power and authority. There are those who are elected lord mayors or city council men, or who hold positions of responsibility because they are appointed to or selected for those positions. There are also persons who have power and control for other reasons—because of wealth, personality, wisdom, cunning, political chicanery, superior physical strength, or from other causal factors. In a hospital there is also a power structure, a planned power structure, which can be represented diagrammatically in the form of an administrative chart; and there are power structures built around goodwill, genuine concern for others, personal ambitions, influence, and other forces, both good and bad.

The planned structure of a hospital involves the inherent power of parliament or some other legislative body that makes laws affecting the hospital and appropriates or withholds money for the administration of its programme. The hospital board, appointed or elected, has the power that comes from hiring and firing personnel and from making policies. The hospital administrator has power because of the authority delegated to him to implement the policies made by parliament or by the hospital board. Power does not end there. The individual doctor has the power which comes from the use of his professional knowledge and skill in treating a patient, and which is implicit in his having final responsibility for what happens to a patient. The superintendent of nurses has power over nurses of lesser rank; the nurse has power over a patient when she gives or withholds something that may add to his comfort. The almoner has power over you and me when we are ill in the ward and she arranges, or fails to arrange, for a troublesome, noisy, annoying patient to leave the hospital as soon as it is in his interest to be released.

Systems of Beliefs and Values.

Systems of beliefs and values are always found in every community. They may be well articulated or less well defined. However, if one listens carefully enough, one hears expression of such beliefs and values as the following: "too much government control is bad"; "all children up to twelve, fourteen, sixteen" (or whatever the case may be) "should have equal educational opportunities"; "men are better car drivers than women" (or vice versa); "a house is more important than a car"; and many others which vary from community to community.

A system of beliefs and values may also be found in a hospital community. One that I believe is fundamental in every hospital with acceptable standards is that a patient has an absolute right to the best medical care available. Another is that no professional person neglects his professional duty for any reason whatsoever when this duty affects the welfare of the patient. A whole system of administrative policies and procedures is built up around these two basic beliefs and values. They are powerful forces in a community, these value systems.

Patterns of Interpersonal Relations.

Patterns of interpersonal relations include ways in which people get together, live together, and get along together. Many factors play important roles in these interpersonal relations. For example, patterns of community and the adequacy of these patterns may determine whether relations are good or not so good. Relations are good when people can work together effectively towards a common goal. Despite the many forces operating in a general hospital community which may influence interpersonal relations adversely, always there is one common objective which has the potential power to affect the pattern positively—the concern of everybody for the welfare of the patient.

Discussion.

The analysis of these eight elements inherent in the structure and functions of a community could be elaborated indefinitely. Perhaps there has been sufficient development to give us not only a basis for calling a general hospital a community, but also a chance to begin to see how these elements affect the "life and labour" of the people who reside in the hospital community. Although in a general way all communities include these eight dimensions, when analysed from the point of view of function and structure, and all people are affected by them, nevertheless it remains true that even as every individual differs from every other individual, so every community has its own unique characteristics. The general hospital, too, has peculiar traits that may have special significance when they interact with the factors outlined above. Some of the special characteristics include the following.

1. A large proportion of the population, as compared with that of other types of communities, is transient. In a general hospital patients come, often unexpectedly because of accident or a sudden onslaught of illness, and stay for a relatively short time. When they are in the hospital community, they are very dependent upon the ministrations of the rest of the hospital population, the staff. They ordinarily are uncomfortable, often unhappy, frequently afraid, and they may or may not project their discomfort and struggle against dependence and fears upon the doctor, nurse, almoner, or cook in the kitchen. They never become completely assimilated as do most newcomers to a city or neighbourhood. Indeed, there are reasons for not encouraging complete assimilation, since one of the goals of the staff is to help the patient emigrate as soon as he is able. The lack of feeling of belonging and the feeling of instability and insecurity that come from incomplete assimilation into any community, and the interaction of this with the struggle against all the other discomforts implicit in most illnesses, may well create tensions and strains that not only affect other patients, but that may permeate the atmosphere of the whole hospital community. This tension and strain may be complicated by the urgency and emergency nature of many a staff member's job. In the interest of the patient's welfare, things must be done promptly, efficiently, accurately and always with maximum skill. There is little opportunity for relaxing while at work. That human relations under these conditions may at times become strained would seem to be inevitable.

2. In a general hospital situation both stratification of population and power structures are very pronounced. What this stratification and power structure is has already been described. What it does, particularly when it is not accepted either by members of the staff or by patients, is quite another story.

Perhaps it was relatively easy for a staff member, nurse, almoner, psychologist, dietitian, X-ray technician and the host of ancillary personnel in a hospital to accept a rather rigid hierarchy of control and status when the education and preparation for these particular jobs was practically non-existent, when there was little professional consciousness, and when the doctor alone had competence and skill. As all personnel have gained in knowledge and experience, and as greater emphasis is being placed in democratic societies upon group participation and group control, to say nothing of the emphasis upon the equality of human beings, there may be more questioning of stratification of a given community and greater feeling of frustration when finally one must always recognize the superior status and greater authority of someone else. Yet there is real purpose behind the stratification of the general hospital, and much can be related to the welfare of the acutely ill patient whose condition demands prompt decision and action. Take, for example, the power and status of the doctor. An article which I read recently equated responsibility with freedom. If the doctor must take final responsibility for the life and death of a patient, he must have freedom of action commensurate with that responsibility. That does not, however, preclude getting information, suggestions, or even advice from other members of the staff if such help is in the interest of the patient.

Patients, too, may react against the stratification and power structure of a hospital. The patient of low status and of an independent, or even rebellious, nature may resent any preference, real or imagined, shown any other patient of higher status, and he may struggle against the absolute power over him when he is flat on his back. This may be true when his dependency and independency needs are customarily in normal relation to each other. When they are neurotic, he may use this power as a crutch on the one hand or as something to struggle against excessively on the other.

3. In a general hospital the eight elements contained in the function and structure of any community operate in an extremely complex manner and create many problems of administration. Administration is said to have for its purpose the smooth operation of all parts of an organization. It attempts to create the kind of situation in which all personnel can work to the best advantage, to the end that together they may achieve the goal of the agency. When the administrative machinery breaks down, as machines have a way of doing, the people in the agency cannot work with maximum efficiency. This can be extremely frustrating, particularly when the patient's condition demands prompt and effective action and the pressures of the general hospital community bear down upon the staff.

In the autumn of 1944 the College of Hospital Administrators in the United States undertook the creating and financing of a long-term undertaking which later became the Joint Commission of Education. With the American Hospital Association as co-sponsor, the study of the problems of hospital administration which was made is summarized in a book (Joint Commission of Education, 1948). In it an abstract of not less than five hundred problems of hospital administration is presented. These are listed as follows: (i) problems centring on medical staff; (ii) personnel management; (iii) medical care — extension or improvement of current services; (iv) business and financial management; (v) community relations: public relations; (vi) regional associations; (vii) physical plant and equipment; (viii) financing changes and additions; (ix) the governing board; (x) problems related to type of control or connexion; (xi) legal aspects: incorporation, liability, litigation. Indeed, as was pointed out in the first issue of *The Australian Modern Hospital*, the modern hospital community presents "a very complex problem". Every patient "requires his share of the boiler plant, stores department, kitchen, nurses' home, lecture theatres, research departments, doctor's quarters, operating suites, out-patient department, X-ray department, pathological laboratory, mortuary, switch room, central records department, lifts, stairs, corridors, ward service rooms, and a host of other necessities" (Stephenson, 1949).

Added to all this complex array of requirements is the necessity of standing ready at all times to make the essential adjustments when one of the physicians, in the interest of a patient's increased chances of recovery, wishes to make a change in treatment—a change which may necessitate a shifting about of equipment or some other accommodations. Then, because of a new discovery in medical science, and the right that entitles all patients to the best kind of treatment known, entirely new equipment may have to be installed or a new regime of care inaugurated. Added to this are problems of personality of staff, who, because they are hard driven, exacting in their own efforts, intent on getting a job done, and close to abnormality at all times, may be impatient, critical, irritable and unable to make the best of a bad situation. Truly a hospital community, at least, a general hospital community, might well tax the cooperative capacities of even a group of angels sent from heaven.

Yet the objectives of the general hospital demand that members of the staff work together, that they cooperate as a team. What, then, can the almoner do to help promote this teamwork? How can she contribute to effective interpersonal relations between all members of the hospital staff? This is the focal point, the nexus, of our discussion.

Thus far I have outlined some eight dimensions of functions and structure which apply to all groups of people that band together in what can be designated a community. In these eight dimensions are elements conducive to positive interpersonal relations and to a lack of them. In the general hospital community certain factors are operating which, when interacting with the elements existing in any community, may tend to intensify the hazards to good cooperative teamwork. I have listed these as the tensions and strains inherent in being ill and not fully assimilated into the community, interacting with the stress that comes from being hard driven, exacting and very responsible as an individual staff member; the pronounced stratification and power structure of the hospital community which, though designed to serve the interest of the patient, may not be accepted by all residents of the hospital community—indeed, may be definitely resisted; and the very complicated nature of the administrative problems which arise in a general hospital situation, and the inevitable breakdown of parts of this administrative machinery at intervals, either frequent or few.

Despite the situations in a general hospital which militate against smooth and easy interpersonal relationships, one cohesive factor exists which, in the final analysis, dominates all else — the absolute belief in the values attached to the sacredness of human life. Out of this grows the patient's inalienable right to the very best treatment available; out of this also stems the dedication of every professional staff member to the cause of the patient's best interests. As has been indicated before, this goal has first place in everything that is done or left undone. The goal is clear and it is wanted at all times. The ways of realizing this goal may, however, be confused.

On another occasion in Sydney I referred to the contribution of one of our American social psychologists, Sherif. Through various carefully planned and conducted research projects, Sherif has demonstrated the importance of a clearly defined and accepted goal in group relationships. Such goals may not only resolve conflicts and other problems of group behaviour, they may prevent them (Sherif and Sherif, 1956).

When this point was discussed with an Australian almoner she said: "Yes, indeed, this one factor is the key to cooperation in a hospital community, given adequate knowledge, adequate skill, and adequate professional self-discipline."

And so it is. The goal by itself is not going to serve optimally its cohesive purpose in a group unless there are adequate knowledge, adequate skill and adequate professional self-discipline. In the case of the almoner, what does this involve?

I shall omit references to how this relates to cooperation between patient and almoner, and also to how conceivably the almoner may contribute to the constructive relationships between the patient and members of other disciplines. These subjects have been discussed at some length in your educational experiences of all kinds, and they have also been developed in the medical social work literature. Instead, I will dwell on the almoner's potential contribution to the cooperative relationships between staff and all members of the hospital community and the hospital administration.

Cooperation between people first of all has to be based upon acceptance of and respect for each other. First of all the almoner must be accepted as a person. One of my colleagues, an American medical social worker in a general hospital, puts it thus:

> I firmly believe that we social workers, not a whit less than others offering goods or services, have to sell ourselves before (or at least along with) our products . . . No amount of professional skill and knowledge will shine through if the social worker has not learned how to be *persona grata* with his colleagues. Further, I believe that very effective "cooperating" has been done when the social worker may have been a bit short on technique but long on acceptance as a person.

I would agree completely with this point of view. Nevertheless, I also believe that in a hospital community, perhaps more than in any other community, respect is fostered by the obvious competence of another staff member—the kind of competence that grows out of "adequate" knowledge and skill. Yes, indeed, professional people in a hospital community—and this goes for many patients too—are "quality conscious". Perhaps the rigorous training of many of the staff has made this so; but whatever the reason, it behoves any member of the staff, including the almoner, to be aware, very acutely aware, of all opportunities for improvement of knowledge and skill as these pertain to a particular job. It does not take long for other members of a hospital staff, including the hospital administrator himself, to sense when an almoner knows what she is doing and why she is doing it, and when she is befuddled in her thinking and unsure of herself. I am not speaking of any forced outward signs of self-assurance, but the kind that comes from within. My experience with doctors, nurses and other staff members of hospitals, who have questioned the value of the contributions of a social worker in a medical setting, has invariably shown that what has won them over was the sureness and sensitivity of touch with which an almoner approached her job, usually in relation to one particular patient in whom the doctor, nurse or other staff member had a special interest.

The almoner also requires knowledge concerning the whys and wherefores of the particular operations of the hospital in which she is employed. She may not always agree with the policies, or agree with the ways in which they are implemented; but she needs to know what they are and why they are. If she has constructive suggestions for improvement, she should make them through proper channels and not be too disappointed if they are not put into practice immediately or ever. Her social work experience should make her realize that advice, as such, whether sought or unsought, is seldom wanted, and if she is in the position of consultant, her recommendation may be accepted or not accepted. Such, alas, are the roles of adviser and/or consultant. However, what does happen is that out of the questions raised by such contributions of a staff member, if they are objectively made, changes which occur may be even better than the original recommendation.

The "adequate" use of professional self-discipline must also have the ingredients of "adequate" knowledge and skill. As a case-worker, the almoner has learned much about the exercise of professional self-discipline in relation to a patient or client. The same dynamics as operate in a patient's behaviour tend to operate in varying degrees in our own behaviour, in the behaviour of our colleagues and in the behaviour of the hospital administrator. The same exercise of objectivity, of timing, of support, of defining issues, of enabling each other to make choices *et cetera*, applies. In discussing the responsibility for promoting cooperative relationships between attorneys and social workers, one of my lawyer friends has repeatedly said to me that the social worker must assume major responsibility. She must go farther than the proverbial half-mile. This attorney bases his case on two arguments (which may or may not stand up in court, but they sound logical): (i) the social worker has had more opportunity to learn what goes into human behaviour than has the average lawyer; (ii) the social worker actually has preempted a field of activity which at one time belonged, in part at least, to the family lawyer. In other words, we are "poaching upon" the attorneys' "preserves". Perhaps many of our colleagues in the general hospital have had as much opportunity as we have had to learn about human relations; but it is true that many of the services now performed by the almoner were originally the responsibility of the doctor and nurse, and in a way we are preempting their fields. Too much stress can be placed on "going the second mile", because in a team relationship there has to be something approaching an equal investment of energy and effort. Nevertheless, we should be very sure that we "carry our own weight" and even go beyond when that is indicated.

So far we have considered the acceptable personality ingredient, the application of adequate knowledge and skill and the use of professional self-discipline, primarily in terms of the relationship of the almoner to other members of the hospital community on an individual basis. The almoner also has many opportunities to play a role as a member of a group, both formally and informally organized.

To me, one of the most illuminating and useful findings that has come out of the recent studies of group behaviour (which, I may add parenthetically, are well summarized by Warren H. Schmidt, 1955) is that in order to contribute to the leadership role in a group, a group member does not have to be the leader, That is, he or she does not have to be in the position of chairman or in the role of leadership because of influence or personality qualities or for other reasons. In other words, leadership can be shared in a group. Schmidt points out that "studies have shown that leadership can be defined in terms of functions to be performed by a group". These functions have been classified into roles. These roles in a group discussion, or perhaps staffing of a case, include such functions as seeking information, seeking opinion, giving information, giving opinions, elaborating ideas, initiating new thoughts, and coordinating and summarizing discussion. When such roles are played in terms of a sincere and intelligent attempt to reach the goal of the group—perhaps to determine what is the best plan of treatment for a patient—then we perform what might be called a task role, and we contribute to the effective cooperation of a group. If, according to the findings of a study made in Pittsburgh, we get our member roles mixed up and become confused about our objective, we may hamper the cooperative relationships of the group. For example, the Pittsburgh study seems to indicate that in a group discussion in which the goal is the best plan of treatment for a client or patient, all members of the group should make that goal the focal point of discussion. If the almoner takes the opportunity to include some education or propaganda concerning the work of the almoner in general, or the doctor decides in the process to instruct all and sundry present concerning the skill necessary for treatment of a particular condition of the eye, the almoner and the doctor are not contributing to the immediate objective for which the group has come together, either formally or informally, and their activities tend to make for division rather than for cooperation. True, the rest of the staff may learn about medical social work in the staff discussion; but, if so, this learning comes as a by-product. There are, or should be, a time and place for the education of all staff members concerning the work of the

almoner, and also concerning something of what goes into the successful treatment of the eye of a patient; but the time and place are not a staff meeting for the purpose of planning the treatment regime of a particular patient.

Another way in which the individual almoner, as a member of a group, can participate in the leadership function is to play the role of morale builder. We help build *esprit de corps* when at the right time and in the proper manner we encourage, support and express the feelings of the group, help the group set standards, call attention to our common goal *et cetera*. Such knowledge of one's role and the exercise of one's role with adequate skill and professional self-discipline make for a feeling of unity and increased freedom for each group member to make his contribution.

My final point is made with some hesitation—with hesitation because it is so much easier to preach than to practise. It also has to do with professional self-discipline and the ability or strength of mind and spirit to exercise this self-discipline. In a community like the general hospital, where tensions tend to run high, there is much room for calm and serene people. The almoner may well concentrate on cultivating calmness and serenity. And how?

I believe each almoner must find her own way and, having found it, must consciously and regularly seek opportunities for replenishing her reservoir of equanimity. Some may find the way through week-ends on the beaches, in the mountains, in the bush, and on horseback. Others may experience a recreation at concerts or through reading; still others may find it through religious activities. For some it may come in a variety of ways; for others in one way; but, if we wish to contribute to a cooperative spirit in a general hospital, come it must.

The almoner, then, can contribute to the cooperative relationships between staff and all members of the hospital community and the hospital administration, by making herself an acceptable and accepted person, by deserving the respect of all colleagues because of her knowledge of and skill in her own field of work, by knowing the whys and wherefores of particular activities, and cooperating in them herself. If she has suggestions for change, she can make them through proper channels and expect her suggestions to be implemented in another form. She can contribute also by going at least half-way in an effort to cooperate with colleagues and basing her cooperation upon her understanding of the dynamics of human behaviour, by playing task role and group maintenance roles with professional self-discipline as she participates in group discussions, and by consciously developing and maintaining a calm and serene spirit. An almoner with the ability to perform in this way contributes to the cooperation in a general hospital community.

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TRANSURETHRAL OR OPEN OPERATION IN PROSTATIC OBSTRUCTION?

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WITH the development of the resectoscope for transurethral resection of the prostate, there have arisen rival claims as to which is the better method for relief of prostatic obstruction—open surgery or transurethral resection. Furthermore there exists a mistaken concept that transurethral resection is necessarily safer.

It has become increasingly obvious that when dealing with an individual patient one cannot generalize about this problem, because each patient has to be judged with regard to his general condition and the type of prostate before the ideal procedure can be instituted. Experience has shown that the fibrous type of prostate causing bladder-neck obstruction is ideally suitable for transurethral resection, whilst the very large vascular prostate is in the main unsuitable. There are exceptions to the latter rule. In prostatic surgery the ultimate aim is a live patient who can pass urine normally, and who will remain this way. Both open surgery and transurethral resection can achieve this aim if the patients are carefully selected. The purpose of this paper is to clarify the correct selection of cases for either method, and to set out the advantages and disadvantages.

For many surgeons who undertake prostatic surgery there is no choice in the matter, because they have not mastered the art of transurethral resection, which takes a lengthy period of apprenticeship with persons proficient in this method. Both methods have disadvantages, which are determined by the type of prostate and the general risk of operation in the individual patient. In many cases personal preference by the surgeon will be the deciding factor. For instance, there is a variance in the figures of individual urological surgeons relating to which method is used. An approximate overall average in Melbourne amongst urologists is as follows: transurethral resection, 65%; open operation, 35%. The significance of this ratio is doubtful, as there has often been a selection before the patient is examined by the resectionist.

Disadvantages of Open Operation.

General anæsthesia is necessary. Although this is becoming increasingly safe, it is not considered to be as safe as low spinal anæsthesia in the elderly subject, with poor respiratory and cardiac reserves, whilst local anæsthesia, in my opinion, is unsatisfactory. It is found that the low spinal anæsthetic anæsthetizes the urethra and prostate satisfactorily for transurethral resection, but not the abdominal wall and bladder. A high spinal anæsthetic, which would be necessary for the open method, lowers the blood pressure.

There is frequently a sudden lowering of the blood pressure during enucleation, due in the main to the brisk bleeding which occurs. In transurethral resection this is prevented by coagulation during the procedure, and there is no sudden lowering of the blood pressure. Thus, for patients considered to be "poor risks", one tends to favour transurethral resection. After open operation, the postoperative period is uncomfortable, and there is a predisposition to wound infection, fistula and hernia. The period in hospital and convalescence are comparatively lengthy. From the point of view of the type of prostate, open operation is unsuitable for fibrous glands, and for carcinomata which are not responding satisfactorily to hormone therapy. Transurethral resection is ideally suited to these cases. Radical prostatectomy for carcinoma is justified only if it can be diagnosed beyond reasonable doubt that the carcinoma is localized in the gland.

Mr. A., aged sixty-two years, presented with acute retention of urine four years ago. His bladder was catheterized and continuous drainage was carried out for two weeks; a clinical diagnosis of carcinoma of the prostate was made and he was given appropriate doses of stilbœstrol. As he was still unable to pass urine when the catheter was removed, he underwent a transurethral resection. The biopsy revealed carcinoma. His convalescence was uneventful and he was given five milligrammes of stilbœstrol three times a day and reviewed at three-monthly intervals. Eighteen months later he reported increasing difficulty with micturition, and hæmaturia. Excretion pyelography revealed good renal function, but post-micturition films of the bladder area showed a fair amount of residual urine. The stilbœstrol dosage was increased to 10 milligrammes three times a day, and there was some improvement over the next six months. In the succeeding twelve months he noticed further increasing difficulty and occasional hæmaturia. Panendoscopic examination revealed multiple nodules of prostatic carcinoma invading the base of the bladder, and the prostatic urethra was partially obstructed. Further transurethral

resection was undertaken, and the dose of stilbœstrol was increased to 15 milligrammes three times a day. Convalescence was uneventful, and since then he has been passing urine normally.

If open prostatic operation is undertaken for the fibrous gland, there may result capsular tears, incontinence of urine and persistent suprapubic fistula. In the last-mentioned case, catheter drainage will allow the fistula to close, but it often breaks down again when the catheter is removed, owing to the presence of residual fibrous prostate causing obstruction at the bladder neck. Subsequent transurethral resection is often necessary. If a small prostate is diagnosed pre-operatively, "wedge resection" of the bladder neck as carried out by suprapubic methods is an unjustifiable procedure when it can be dealt with endoscopically.

Mr. B., aged sixty-five years, presented to his surgeon with a history of increasing dysuria and nocturia. Investigation showed that he had a small, firm gland causing bladder-neck obstruction. A difficult suprapubic enucleation with excision of the prostatic tissue was undertaken by the Freyer method, and the bladder was drained suprapubically because of hæmorrhage. After five weeks in hospital, with episodes of drainage with an indwelling catheter, the suprapubic fistula persisted. After a transurethral resection of a small amount of remaining tissue the fistula closed and remained closed after five days of post-resection catheter drainage. The patient is now passing urine normally.

Suprapubic prostatectomy requires a longer stay in hospital, particularly if post-operative suprapubic drainage is used. In the closed bladder type of operation—for example, the Harris and Millin types—in my hands the period in hospital is similar to that for transurethral resection. In the neurogenic cases with retention of urine —for example, in paraplegia, disseminated sclerosis, tabes, and post-abdomino-perineal rectal resection, open operation is undesirable, whereas limited transurethral resection, repeated if necessary, may suffice to overcome the retention.

Disadvantages of Transurethral Resection.

Instrumentation of the urethra with the resectoscope carries with it the risk of stricture formation. Although this can be reduced by the use of the multiple sheath instrument, meatotomy, perineal urethrotomy and resecting for the shortest possible time, it is nevertheless a calculated risk. However, urethral stricture also occurs after open operation from catheterization and infection.

It has been found by experience that the operative hazards increase sharply if a resection takes more than one hour. If it is apparent when assessing a patient for relief of his bladder neck obstruction that a complete resection will take more than one hour, then it is reasonable to think in terms of open prostatectomy. If, on the other hand, the patient is a very poor risk and has a large gland, then an incomplete resection, repeated if necessary, will often suffice for the remaining period of his life. I have adopted this principle in the care of five recent patients with acute retention of urine who were considered very poor risks for open operation and who had large vascular glands. In two of these cases I resected tissue around the whole circumference of the prostate and was troubled with bleeding. In the others only one lobe was resected, the rest being left intact. Consequently there was less surface from which to bleed, the risk being thereby diminished. Although these patients were able to pass urine satisfactorily after the removal of the catheter inserted after operation, one can expect further obstructive attacks, but these can be dealt with as they arise. The important point is that these patients are alive and passing urine.

If the smaller sheaths of the resectoscope are used it takes longer to resect the same amount of tissue, because of limitation in the cutting loop size and the difficulty of extracting large pieces through a small sheath. Consequently transurethral resection of a large type prostate is not undertaken unless the urethra will accommodate the 28 French sheath.

In the medium to large size prostate, in the good risk or younger patient, it is thought that open operation of the Millin type gives better end results. There is less risk of post-operative stricture and it is less likely to recur.

One of the more serious disadvantages of transurethral resection is that intravascular hæmolysis can occur, and this may be fatal. Fortunately this is rare. In over 300 consecutive cases of transurethral resection with which I have been associated, there has been one fatal case due to intravascular hæmolysis and two cases in which mild transient jaundice developed. This risk is allegedly reduced when glycine (1% solution) rather than water is used for irrigation during and after the procedure.

From the point of view of geographical consideration, open operation is frequently more convenient for the patient and visiting relatives. The performance of transurethral resection requires a valve diathermy unit for clean cutting. This is expensive and practically non-transportable and is consequently not available at most of the smaller hospitals. This practical fact usually necessitates city treatment for country patients undergoing transurethral resection.

If there are associated bladder conditions, such as a large stone or tumour, transurethral resection may be contraindicated because of the increased operating time taken to deal with such lesions endoscopically. If the bladder is opened, these can be readily dealt with before the proposed prostatectomy. The presence of an associated diverticulum rarely influences my decision away from transurethral resection, because many diverticula are symptomless when the obstruction is removed. If necessary a narrow-necked diverticulum can be enlarged with the resectoscope and given the chance to drain more freely.

Discussion.

It will be seen from this discussion on the disadvantages of both methods that one frequently has to arrive at a compromise when assessing the procedure better suited to the individual patient.

I believe that it is a mistaken concept that transurethral resection is necessarily the safer procedure. It is safer for some patients, but not for all. For example, for patients with large vascular glands, transurethral resection may have to be abandoned at the time and open operation performed because of bleeding, which cannot be adequately controlled by diathermy, and which obscures the telescopic vision. With careful selection this type of accident can be avoided.

Selection of Patients.

There are two main features in the selection of patients which deserve consideration—first the medical condition of the patient himself, and secondly, the type of prostate causing the obstruction. With regard to the medical condition, the pertinent question is, will the patient stand a general anæsthetic and a lowering of the blood pressure? In many cases one can only guess at this, and in the doubtful cases I am guided by the opinion of a physician, who is better able to assess such conditions as hypertension, heart failure, diabetes, uræmia and anæmia, to mention some of the common associated disorders. Renal function tests and an excretion pyelogram are informative in the assessment.

If it is decided that open operation is too risky for such patients, then one is practically forced into a resection or suprapubic cystostomy under local anæsthesia—this being undesirable in most cases.

In the assessment of the type of prostate, one is guided by the broad classification of small and large. There is no doubt that small glands are better resected. The difficulty lies in our inability always to assess accurately the size of the prostate with a finger in the rectum. A small fibrous prostate may feel large when the bladder is distended, or if there has been recent acute retention of urine. This is due to œdema and swelling within the gland, and it will subside after a few days' catheter drainage and perhaps the administration of sulphonamide or antibiotics.

Mr. C., aged sixty-seven years, was admitted to hospital two years ago with acute retention of urine. He had a painful bladder, the fundus of which was palpable at the

umbilicus. There had previously been minimal urinary symptoms. I thought his general condition was good, and on rectal palpation could feel a smooth globular prostatic enlargement, which was classified at the time as a Grade II benign gland, suitable for a Millin's prostatectomy. At that time I was anxious to complete a series of "immediate prostatectomies"; but in this patient I detected some cardiac irregularity. A physician was called in consultation with regard to the heart condition and he was of the opinion that operation should be deferred for some weeks until the cardiac condition had improved. For this reason a cystostomy was established under local anæsthesia. During the post-operative period the following investigations were carried out. An electrocardiogram showed auricular fibrillation. An excretion pyelogram was essentially normal, with good concentration of the dye. There were shadows suggestive of metastases in the bones of the pelvis. The serum acid phosphatase level was 12 units.

After five days' suprapubic drainage a further rectal examination disclosed that the prostate was now very small and firm and there was a small nodule in the right lobe. He was given five milligrammes of stilbœstrol three times daily and after two weeks he was passing urine satisfactorily with the suprapubic tube clipped off. After a further week this was removed and the sinus had closed within a fortnight. He is now well and passing urine satisfactorily on a maintenance dose of five milligrammes of stilbœstrol twice daily.

The most interesting single feature about this case was the way in which the prostate diminished in size with bladder drainage.

A thick capsule will give the impression of a gland much larger than it is. On several occasions when I have enucleated a prostate which I had thought by rectal palpation alone to be too big to resect adequately, I have been dismayed at the small size of the enucleated adenoma, whilst there has been a very thick capsule. On the other hand, a gland may feel small and yet have a large intravesical enlargement not readily palpable. Bimanual palpation will usually enable an accurate estimation of the size and mobility of the prostate to be made; but the most reliable means of accurate assessment is panendoscopy with the fore-oblique telescope. The cystoscope with its right-angled vision is misleading in assessing the size. Unfortunately instrumentation may precipitate acute retention of urine in patients selected for interval operation. Consequently, in patients selected for panendoscopy for assessment as to whether the prostate is resectable or better removed by open methods, a hospital booking for about two weeks is desirable.

The method which I have found satisfactory in doubtful cases is to have both endoscopic and "open" instruments available at the panendoscopic examination. If it is found that the prostate is adequately resectable, then resection is proceeded with; if not, and if the patient has been assessed as a good risk, then the open method is employed forthwith. In the case of a poor risk, a limited resection is undertaken or a cystostomy is established.

Summary.

1. The indications for transurethral resection or open prostatic operation in individual patients are discussed, together with the disadvantages of each procedure.

2. The facts that open prostatic operation is reserved for the good risk patient with a large vascular benign gland, and that transurethral resection is preferable in most other cases, are discussed.

3. The method of assessing the type of prostate causing the obstruction, and the pitfalls in this assessment, are considered.

Acknowledgements.

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Reports of Cases.

GANGLIOGLIOMA OF THE BRAIN STEM.

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TUMOURS of which neurons form an integral part may occur within the central nervous system. A clear description of such a tumour was published over fifty years ago by Worcester (1901), and in 1941 Courville and Anderson were able to collect 27 cases from the literature. However, in many text-books of pathology the tumours are not mentioned.

Only one report of a nerve-cell tumour of the central nervous system has appeared in the Australian literature (Cox, 1932).

Clinical Record.

A boy, aged seven years, was admitted to the Royal Children's Hospital, Melbourne, in July, 1955. His mother said that from the age of eighteen months he had vomited almost every morning, although in other respects he had remained well until he was four years old. At that time sudden onset of difficulty in swallowing and weakness of his left shoulder and left leg led to his admission to hospital with a diagnosis of poliomyelitis. Partial recovery occurred during the following month, but some weakness of the shoulder and leg persisted. At the same time a squint of the left eye was noticed, and one year later this was corrected surgically. Difficulty in swallowing recurred when he was aged six and a half years, accompanied by recurrence of the squint and by weakness of the left side of the face. His disabilities slowly increased in severity, and his admission to the Royal Children's Hospital was precipitated by the onset of fever, with cough and respiratory difficulty.

Examination of the patient revealed him to be a well-nourished boy of slight build. He was drowsy, but easily aroused and quite cooperative. The head circumference was 53 centimetres. His gait was unsteady, and he tended to fall toward the left. Power and tone seemed normal and the deep reflexes equally active in all limbs, but left ankle clonus was elicited. There was a bilateral Babinski plantar response. The pupils were small, reacting normally to light and to accommodation. The ocular fundi appeared normal. Conjugate deviation of the eyes to the left was impaired, as was nasal deviation of the left eye. There was left facial weakness, and the tongue, when protruded, deviated to the right. Pooled saliva was present in the pharynx. The left vocal cord was immobile. No abnormalities of sensation were detected. The blood pressure was 160 millimetres of mercury, systolic, and 100 millimetres, diastolic. Signs of bronchopneumonia were present, but general systemic examination revealed no other abnormality. He died on the day after his admission to hospital.

Post-Mortem Examination.

The calvarium, meninges and cerebral hemispheres appeared normal. The brain stem was distorted by a mass which expanded the left side of the medulla and pons, displacing the vertebral arteries and the first part of the basilar artery to the right. The surface of the cerebellum appeared normal.

Sections were made through the brain in the coronal plane. No abnormalities were detected in the cerebral hemispheres, and the lateral and third ventricles were normal in size and shape. When the brain stem was cut, the cause of its surface deformity was found to be a roughly spherical mass, 2·5 centimetres in diameter, which occupied a part of the left side of the pons and medulla (Figure I). This mass was composed of a firm, almost homogenous grey tissue, clearly demarcated from the surrounding tissue, but not obviously encapsulated.

In the pons the rounded mass was situated mainly in the left side of the tegmentum. Laterally it displaced outward the left *brachium pontis* and appeared to invade it. Medially it displaced the fourth ventricle toward the right, and at the ponto-medullary junction the ventricle was reduced to an antero-posteriorly directed slit. The mass extended caudally about five millimetres into the medulla, involving a part of the restiform body on the left side. The grey tissue thus occupied only a part of the greatly expanded left side of the medulla, the remainder being occupied by a homogeneous white tissue of firm consistency in which few anatomical outlines could be distinguished. The expansion ended quite suddenly in the caudal end of the medulla, the cord being unaffected.

Apart from bronchopneumonia, examination of the other systems revealed no abnormality.

Microscopic Examination.

Sections taken from different parts of the tumour were similar in appearance. The tissue was very cellular (Figures II and III), consisting of numerous large oval cells, supported singly and in small groups by mature glia of moderate vascularity. Many groups of small round

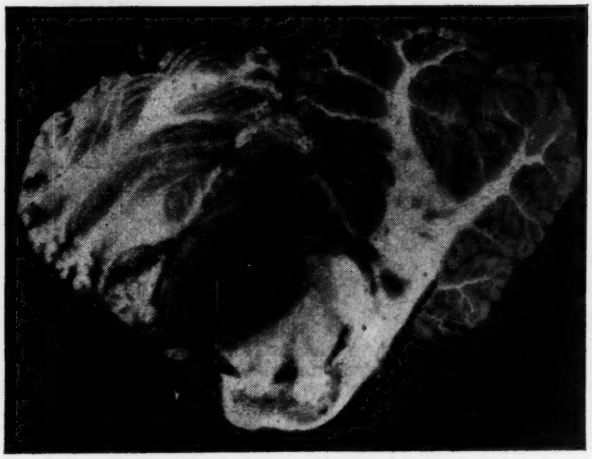

FIGURE I.

Section through cerebellum and pons, showing tumour in left side of pons.

cells and plasma cells were present. There was no capsule, but the tumour was sharply delineated from the surrounding tissues.

The large oval cells had the appearance of neurons. They varied greatly in size, but the majority measured about 30μ by 26μ in diameter. Their abundant cytoplasm was finely granular, with crescentic accumulations of Nissl substance toward the periphery of many cells. Intracellular neurofibrils and occasional clear cytoplasmic vacuoles were present. The nuclei were round or oval, with a prominent nuclear membrane, open chromatin net and a single, deeply staining nucleolus.

Occasional large binucleated cells were seen, but there were no mitotic figures. The number of processes possessed by these neurons varied greatly. Most appeared to have none, but a few had many dendrites and a characteristic axon which could be traced for only a short distance. Occasional nerve fibres could be distinguished between the cells. The tissue supporting the neurons was predominantly neuroglial, the glial elements appearing quite mature. Connective tissue was confined to the immediate vicinity of the blood vessels. Groups of small round cells and plasma cells were aggregated particularly about the vessels, but were present in varying numbers throughout the tumour.

The pale tissue surrounding the tumour was found to be mature glia, in which only occasional neurons were seen. These were considered to be normal cells included in a reactive gliosis rather than neoplastic elements.

Discussion.

Although nerve cell tumours of the central nervous system are uncommon, they have characteristic pathological features distinguishing them from other tumours of the region.

They are benign tumours usually occurring in the second and third decades of life. There is no predominant sex incidence. They are usually situated in the floor or lateral walls of the third ventricle, but they may also be seen in the cortical or subcortical regions of the cerebrum or cerebellum, in the pons or in the medulla. They have been found also in the cervical part of the spinal cord.

The gross appearance of the tumour is usually that of a firm round mass, well demarcated from the surrounding tissue. The colour varies, but is usually grey or pink. Cysts of varying size may be present.

The characteristic histological feature is the presence of mature or immature ganglion cells. The glial elements may also vary in maturity, and the amount of connective tissue associated with the blood vessels has differed greatly in the reported cases. Vascularity likewise varies, and calcospherites are frequently seen. Collections of small round cells, usually situated around the blood vessels, have been present in more than half the reported cases. Wolf and Morton (1937) studied these cells and concluded that they were lymphocytes and not "neuroblasts" or "medulloblasts" as had been thought by earlier workers.

A special form of this tumour occurs rarely as a diffuse growth, particularly in the cerebellar cortex.

The question of terminology has been discussed by almost every writer in this field, and a great number of names have been proposed. Wolf and Morton reviewed 50 cases, which they divided on a histological basis into five differently named groups. Courville and Anderson (1941) did not support this classification. They considered that the various groups showed no relation to anatomical site or to survival time, and that the classification was of no clinical value and was unsound histologically. They concluded that the term ganglioglioma should be used for all nerve cell tumours of the central nervous system.

Scherer (1940) points out that normal neurons may be included within an infiltrating glioma, and such a tumour must not be classified as a ganglioglioma. Failure to make this distinction may account for the high incidence of gangliogliomata reported by some pathologists.

Summary.

Gangliogliomata are benign tumours consisting of nerve cells and glial tissue occurring within the central nervous system. Clinical and post-mortem records of a boy in whom such a tumour occurred are presented.

The gross and microscopic appearances of these tumours are briefly described.

Acknowledgements.

I wish to thank Dr. L. P. Wait and Dr. E. K. Turner for permission to publish this case. Dr. J. W. Perry and Dr. A. L. Williams supplied helpful criticism and advice. The photomicrographs were prepared by Dr. A. L. Williams, and the photograph by Mr. C. Murphy, of the photographic department, Royal Children's Hospital.

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TWO CASES OF MYOCARDITIS ASSOCIATED WITH PHENYLBUTAZONE THERAPY.

By P. R. HODGE AND J. R. LAWRENCE.
From the Department of Pathology, University of Adelaide.

SINCE the introduction of phenylbutazone ("Butazolidin") therapy in 1952, numerous reports of the toxic effects of the drug have appeared. Those more commonly reported include skin rashes, blood dyscrasias, œdema and gastro-intestinal hæmorrhages. These have been summarized by Stephens *et alii* (1952), by Kuzell *et alii* (1952), and in *The Lancet* (symposium, 1953). In Australia, Kelly (1954) has reported a similar series. Other less common effects include hepatitis (Engleman *et alii*, 1954, and McCarthy and Jackson, 1955) and myocarditis (Engleman *et alii*, 1953, and Steinberg *et alii*, 1953).

Case I.

A housewife, aged thirty-eight years, the mother of three children, complained of backache. An X-ray examination of her spine revealed osteophytic lipping, and on March 13, 1956, she was given phenylbutazone, 200 milligrammes thrice daily, and *Tabellæ Codeinæ Compositæ* for seven days, with great subjective improvement. The dosage was then reduced to 400 milligrammes daily. One week later the dose was 200 milligrammes daily, but some ten days after this she did not feel well and discontinued taking the tablets. By April 4 the patient was anorexic and vomiting, her temperature was 103° F., and she had a tender, palpable liver with bile and urobilinogen in the urine. Six days later she developed a diffuse rash, and in three days her extremities had become œdematous.

She was admitted to the Northfield Wards of the Royal Adelaide Hospital on April 24, where she was found to be moderately jaundiced, with a widespread erythemato-macular rash and swollen fingers, eyelids and feet. There were tender enlarged glands in the neck, axillæ and groins, with considerable hepatomegaly, and the spleen was palpable.

The following investigations were then performed. Examination of the blood showed that the hæmoglobin value was 14·3 grammes *per centum*; the leucocytes numbered 17,100 per cubic millimetre, 43% being neutrophile cells, 50% lymphocytes and 7% monocytes. Liver function tests gave the following results. The icteric index was 25 units, the colloidal gold test result was "5", the cephalin cholesterol test produced a "+++" result, and the zinc sulphate turbidity was 23 units. The serum alkaline phosphatase content was 62 King-Armstrong units. The patient was treated with "Anthisan", penicillin and ascorbic acid. By May 12 her condition had greatly improved and there was no jaundice or rash; but the urine still contained some urobilinogen. She was discharged from hospital to convalesce at home.

One week later, having apparently continued to improve, she began to feel tired, vomited and felt breathless with retrosternal "tightness". When she was readmitted to hospital on May 22 she had tachycardia, very faint heart sounds and an unrecordable blood pressure. The liver was enlarged to the level of the umbilicus. She died some twenty-four hours after her admission. The electrocardiograph report was as follows:

> The striking features are the low amplitude throughout the tracing, the extent and degree of *S-T* segment elevation in leads 1, 2, aVL and V2, 3, 4, 5, 6 and 7, and the absence of *Q* waves. The E.C.G. indicates extensive myocardial damage which is not distributed in a fashion one can relate to the occlusion of one or more coronary arteries and yet suggests more extensive subepicardial injury than is usually seen in diffuse myocarditis with pericarditis.

The tracing is shown in Figure I.

At autopsy the relevant macroscopic findings were as follows. Generalized filmy pleural adhesions were present. There was excess straw-coloured pericardial fluid, and the surface of the heart, particularly over the right ventricle and most densely over the auricular appendages (Figure II), was studded with discrete and confluent white, smooth, round elevations up to two millimetres in diameter. In a small area posteriorly the myocardium was hæmorrhagic, and examination of the cut surface revealed diffuse pale streaks, which were also visible from the endocardial surface. There were no granulations on the valves. The spleen was enlarged and firm and the liver was considerably enlarged. The cut surface of the liver was congested with scattered discrete, white, mottled areas up to two millimetres in diameter. The kidneys were congested, but no other gross abnormality was noted.

The microscopic findings were as follows. The heart was affected by diffuse interstitial myocarditis with very extensive destruction of muscle (Figure III). The inflammatory infiltrate consisted mainly of plasma cells, lymphocytes and large macrophages with numbers of eosinophile cells. Some multinucleate giant cells were present (Figure IV). In the small arteries there were early fibrinoid degenerative changes, most pronounced in the media. In the liver there were scattered small granulomata, most pronounced in the portal tracts, with mild centrilobular degeneration. The granulomata were small circular collections of large mononuclear cells. Examination of the kidney, spleen, paraaortic nodes and lung revealed occasional perivascular granulomata. Those in the kidney particularly were similar to "healing" *polyarteritis nodosa*.

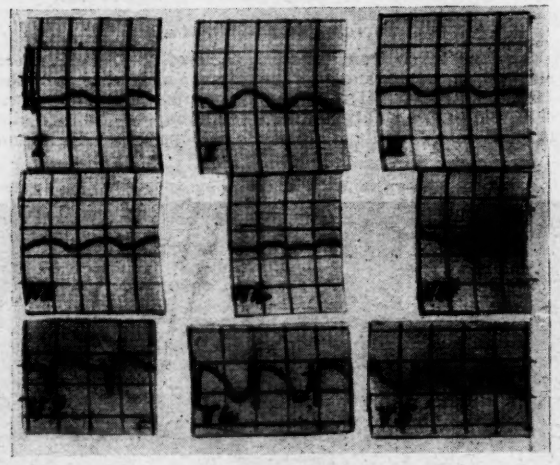

FIGURE I.
Electrocardiogram, Case I.

Case II.

The patient, a widow, aged seventy years, had been receiving phenylbutazone for three weeks for osteoarthritis. The dose given was 600 milligrammes daily, reduced to 400 milligrammes after three days, then 200 milligrammes daily for the last two weeks. One week prior to her admission to the Royal Adelaide Hospital she developed generalized *erythema multiforme* with associated pyrexia. Three days later she developed bronchitis, for which she received "Sulphatriad", four grammes daily. Examination of the patient on her admission to hospital revealed some hypertension (blood pressure 185 millimetres of mercury, systolic, and 125 millimetres, diastolic), with generalized *erythema multiforme* and mild bronchospasm. On the following day the patient developed *status asthmaticus*, in which she died despite treatment.

At autopsy the rash was still present on the face, neck, arms, back and legs. The heart was of normal size and macroscopic examination of the valves, myocardium and coronary vessels revealed no abnormality. In the lungs there were areas of collapse with dependent congestion and some associated œdema. The liver was normal, but the kidneys were rather small and granular. No abnor-

mality was detected in the remaining organs apart from diverticulosis of the descending and sigmoid colon.

Microscopic examination of sections of the heart muscle revealed multiple focal perivascular granulomata comprising macrophages, acute inflammatory cells, eosinophile cells and occasional giant cells. Linear streaks of similar inflammatory cells were present between muscle bundles (Figure V). Further granulomatous lesions were present in the parenchyma and periportal areas of the liver, although in this case there was more pronounced reactive fibrosis (Figure VI). In the sections of skin there were further granulomatous foci immediately beneath the epidermis and within the dermis.

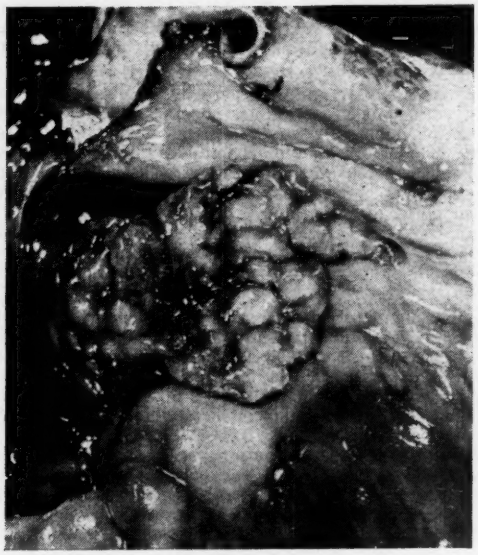

FIGURE II.

The anterior aspect of the base of the heart, showing granulomata in the auricular appendage.

Discussion.

Few instances of a severe myocardial lesion accompanying phenylbutazone therapy have been reported. Steinberg *et alii* (1953) described a perivascular granulomatous myocardial lesion in a subject with associated agranulocytosis. Engleman *et alii* (1953) reported "profound interstitial myocarditis" and associated changes in the liver and kidney. MacCarthy and Jackson (1955) found centrilobular hepatic necrosis with granulomatous focal myocarditis.

The lesions are similar to those previously described in sulphonamide toxicity (More *et alii*, 1946), and to those seen as part of a generalized non-specific hypersensitivity response to a variety of agents (Churg and Strauss, 1951).

Because of this lack of specificity it is difficult to prove the ætiological significance of any given factor; but in view of the foregoing reports and the absence of any other probable causative agent, we consider that Case I could reasonably be attributed to phenylbutazone. Case II is complicated by the history of sulphonamide therapy; but the fever and rash (recognized hypersensitivity manifestations) were both present before the brief period of "Sulphatriad" therapy began.

The occurrence of these reactions in patients who have not been receiving excessive doses supports the hypothesis of hypersensitivity. Both patients were carefully supervised and their dosage had been reduced to 200 milligrammes daily more than a week before "toxic" effects appeared. Most reports suggest that the lesions will be apparent in the first month or six weeks of therapy.

One of us (P.R.H.) has seen focal granulomata in the myocardium of a further small number of subjects who died from agranulocytosis or bleeding peptic ulcer attributed to phenylbutazone. The relative frequency of this lesion and the severity of the myocarditis compared with the lesions in other organs in both cases are in accord with Steinberg's early observation that in his case there was more extensive myocardial than renal or other visceral damage. This is in contrast to the usual effects of sulphonamides.

At autopsy the principal hepatic lesions in each case were focal granulomata situated mainly in the portal tracts. However, further diffuse parenchymal damage has been reported by Engleman *et alii* and by MacCarthy and Jackson. They describe changes varying from "acute toxic hepatitis" to ultimate portal cirrhosis. In Case I there was clinical evidence of severe liver damage four weeks prior to death. Post-mortem examination of sections suggests that the parenchymal damage had largely healed.

While acknowledging the great value of this antirheumatic drug, and realizing that sulphonamide therapy has not been abandoned because it too may produce similar lesions, we believe that the warning given in *The Lancet* editorial of 1953 remains true:

> In these circumstances, Phenylbutazone should not be used merely as an alternative analgesic in rheumatism. Careful selection of patients and close attention to signs of toxicity are rightly demanded.

Summary.

Two cases of myocarditis proven at autopsy in patients who had received phenylbutazone therapy are described. Further granulomatous lesions were present in other viscera. Evidence is presented that the lesion is a hypersensitivity response.

Acknowledgements.

The authors are indebted to Professor J. S. Robertson for his help and advice in the preparation of this article, and to Dr. J. M. McPhie for interpreting the electrocardiogram.

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A CASE OF SICKLE CELL ANÆMIA.

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SICKLE CELL ANÆMIA is an uncommon disorder almost entirely confined to the Negro race. On rare occasions it has been reported in Greeks, Italians and Sicilians, and in certain primitive hill tribes in India. In this disorder

there is an inherited abnormality of the hæmoglobin of the red cells. The presence or absence of this abnormal hæmoglobin, called hæmoglobin S, appears to be determined by a single gene. Sickle cell anæmia will occur in a person who has inherited this abnormal gene from both parents. Hæmoglobin S is particularly insoluble in the reduced state, and tends to crystallize out of solution within the red cells under conditions of reduced oxygen tension. When crystallization occurs, the red cell assumes a sickle-like shape. Occasionally sickle cell anæmia occurs in persons only one of whose parents can be shown to possess the sickle cell trait. In such cases the other parent is usually found to contribute a gene for some other abnormal hæmoglobin or a gene for thalassæmia. Up to the present time the sickle gene appears to have been absent from Australia.

Clinical Record.

The patient, a Greek boy, aged five years, was admitted to the Wollongong District Hospital on April 27, 1956. For the four days prior to his admission to hospital his mother had noticed that he was limping on the left leg, and that he appeared to be suffering from pain when he moved the left leg.

On examination in hospital, the patient was seen to be a thin, unhappy child. His complexion was dark. Passive movement of the left thigh caused pain. There was some limitation of all movements of the left thigh because of the pain induced. Even at rest the patient appeared to experience some pain. His temperature was recorded as 98° F. The pulse was regular and the rate was 90 beats per minute. The blood pressure was 100 millimetres of mercury, systolic, and 60 millimetres, diastolic. The apex beat was felt in the sixth intercostal space 2·5 inches from the sternum. A systolic murmur was heard maximally in the fourth intercostal space just to the left of the sternum. This murmur was propagated into the aortic area and also could be heard in the mitral area. The scleræ were normal. The spleen was not palpable. The liver was palpable one finger's breadth below the right costal margin. It was not considered to be enlarged. Most of the teeth were carious. No other positive findings were noted. There was no ulceration of the legs. The malar bones did not appear unduly prominent.

The patient had in the twelve months before this admission to hospital suffered from attacks of abdominal pain. On three occasions within the past six months he had been taken by his mother to a doctor because of these pains. On each occasion he was admitted to the hospital, where he was observed for a period of approximately ten days. He was then discharged, as the pain had disappeared. On two occasions a diagnosis of possible appendicitis was made. On the other occasion a diagnosis of possible meningitis was made. On each occasion his temperature on his admission to hospital was elevated, but it became normal within a few days. The total white cell count was elevated above 17,500 cells per cubic millimetre on the two occasions on which this test was performed. On his return home his mother noticed that the attacks of abdominal pain continued much as they had before. When the pains occurred, his appetite was poor and he did not sleep well.

The patient is an only child. His parents are apparently of pure Greek stock. His mother and father were born in the same village near the town of Lesbos on the island of Mitilini, approximately 200 miles north-west from Athens. The boy's maternal grandparents live in this same village and are in good health. The boy's maternal grandfather has had treatment for ulcers of the stomach. They have one son and a daughter apart from the boy's mother; both are well. There is no history of anæmia in any of the maternal relatives. On the father's side there are three sisters, and they are well. The paternal grandmother died at the age of seventy-five years and the paternal grandfather died at the age of eighty years. There is no history of anæmia in any of the paternal relatives.

A provisional diagnosis of a possible traumatic lesion to the left thigh was made, even though there was no history of trauma.

A number of special investigations were carried out.

X-ray examinations gave the following findings. No abnormality of the hip joints was detected. Views of the cranial bones revealed thickening of the frontal bones and a slight degree of thickening of the parietal bones. The inner table was not involved. These bones had a laminated appearance, which was more evident in the frontal region. There was apparent filling in of the antra. There was no cardiac enlargement, the lungs appeared clear, and the bones of the thoracic cage were normal apart from congenitally small first ribs.

It was concluded that the changes in the cranial bones and the maxillary regions were indicative of congenital hæmolytic anæmia.

An electrocardiographic examination was made. Apart from sinus tachycardia the electrocardiogram was normal for the patient's age.

The pathological findings were as follows. The hæmoglobin value was 9·0 grammes per 100 millilitres of blood. The white cell count was 13,700 per cubic millimetre, of which 5500 were polymorphonuclear cells, 6000 were lymphocytes, 1200 were monocytes, 800 were eosinophile cells and 200 were basophile cells. Examination of a stained blood film revealed pronounced polychromasia and pronounced anisocytosis. A few elongated crescent-shaped cells were present, together with a moderate number of target cells. A very occasional nucleated red cell was present.

Further hæmatological investigations yielded the following results. Reticulocytes numbered 405,000 per cubic millimetre. The red cells numbered 2,900,000 per cubic millimetre. The mean corpuscular volume was 104 cubic microns, the mean corpuscular hæmoglobin was 31 micromicrogrammes, and the mean corpuscular hæmoglobin concentration was 33 grammes *per centum*. The sickling phenomenon test was performed. Examination of a sealed "Vaseline" preparation of the patient's blood showed almost 100% sickling of the red cells within four hours (see Figure I). The serum bilirubin content was 2·0 milligrammes per 100 millilitres of serum. The zinc sulphate turbidity was nine units, the normal range being from two to eight units. The serum alkaline phosphatase content was 27 King-Armstrong units. Schlesinger's test for urinary urobilin was performed, and revealed increased urinary urobilin in a twenty-four hour specimen of urine. The alkali-resistant hæmoglobin test was performed, and a result of 10% alkali-resistant hæmoglobin was obtained. The results of the saline osmotic fragility test were as follows: hæmolysis commenced at 0·35% saline (normal control, 0·45%), and was complete at 0·1% saline (normal control, 0·32%). Electrophoretic separation of the hæmoglobin revealed the presence of hæmoglobin S.

A diagnosis of sickle cell anæmia was made. The parents' blood was then investigated, with the following results.

Patient's Mother.

The hæmoglobin value was 12·5 grammes per 100 millilitres, and the reticulocytes were less than 1% of the red cells. No abnormality of the red cell size or shape was noticed in the stained film preparation. The results of the sickling phenomenon test were as follows. Only a very occasional red cell showed sickling in a sealed "Vaseline" preparation examined after twelve hours. The addition of a drop of a reducing agent (2% sodium bisulphite solution) to a drop of blood followed by sealing with "Vaseline" produced pronounced sickling of the red cells within two hours. Electrophoretic separation of the mother's hæmoglobin revealed the presence of hæmoglobin S. There was 1·6% alkali-resistant hæmoglobin detected in the mother's blood.

Patient's Father.

The hæmoglobin value was 17·4 grammes per 100 millilitres of blood, and the reticulocytes were less than 1% of the red cells. No abnormality of the red cell size or shape was noticed. A "Vaseline" sealed preparation of the father's blood produced sickling of the red cells after over-

night incubation. Electrophoretic separation of the father's hæmoglobin revealed the presence of hæmoglobin S.

Discussion.

Sickle cell anæmia in white persons is rare. Dacie (1955) suggests that the origin of the abnormal gene in these cases is often uncertain. Wintrobe (1951) lists 13 cases, mostly in Greeks, Italians and Sicilians. He considers that ancestral Negro blood can be suspected in these cases. Castle (1955) believes that sickle cell anæmia in white persons usually occurs in children of parents heterozygous for sickle cell trait and thalassæmia respectively. Neel (1951), however, suggests that cases of homozygous sickle cell anæmia may have been misdiagnosed as sickle cell-thalassæmia disease, because of failure to demonstrate sickling of cells in one of the parents who has the sickle cell trait. According to Silvestroni and Bianco (1952), sickle cell-thalassæmia disease can be distinguished from homozygous sickle cell disease. Sickle cell-thalassæmia disease, according to these authors, is an anæmia of microcytic hypochromic type. Examination of a stained blood film usually reveals many target cells, as well as pronounced anisocytosis and poikilocytosis. Only one of the parents can be shown to have the sickle cell trait. The other parent has the thalassæmia trait. These authors suggest, on the other hand, that homozygous sickle cell disease is an anæmia of normocytic or macrocytic type. The hæmoglobin concentration is usually normal. Moderate anisocytosis is usually present, and a few sickle-shaped cells may be present in the stained blood films. Both parents can be shown to have the sickle cell trait.

The presence of increased amounts of alkali-resistant hæmoglobin and of a decreased saline osmotic fragility of the red cells can occur in both sickle cell-thalassæmia disease and homozygous sickle cell disease.

In the case described the patient has anæmia of macrocytic normochromic type. A few conspicuously elongated cells and an occasional sickle cell could be seen in the stained blood film. The hæmoglobin present in the red cells was shown to be a mixture of types S and F. The parents were each shown to have the sickle cell trait. The findings, therefore, are in favour of the diagnosis of homozygous sickle cell anæmia in this boy.

In view of the large number of migrants arriving in Australia from Greece, Italy and Sicily, it is probable that further cases of sickle cell anæmia in Australia will occur.

Summary.

Sickle cell disease in a Greek boy, aged five years, is described. Both parents were found to have the sickle cell trait. The hæmatological findings were in favour of the diagnosis of homozygous sickle cell anæmia.

Acknowledgements.

I wish to thank Dr. R. J. Walsh, Director of the Red Cross Blood Transfusion Service, Sydney, for his helpful advice in the preparation of this paper. The Red Cross Blood Transfusion Service provided the photomicrograph, confirmed the diagnosis and carried out electrophoretic studies of the hæmoglobins of the boy and his parents. The alkaline denaturation test was performed by Dr. H. K. Kronenberg, of the Fairfax Institute of Pathology, Royal Prince Alfred Hospital, Sydney. The X-ray report was prepared by Dr. W. Lachlan, radiologist at the Wollongong District Hospital, Wollongong, New South Wales.

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Reviews.

Radiology of the Alimentary Tract in Infancy. By Roy Astley, M.B., M.R.C.S., D.M.R.; 1956. London: Edward Arnold (Publishers), Limited. 9¼" × 6", pp. 295, with illustrations. Price: 50s.

THIS book is based on wide personal experience and contains valuable information which can be obtained only in special hospitals. The average specialist in a general hospital has little opportunity to study these cases. The œsophagus is dealt with in detail, and numerous cases of atresia are reported and illustrated. "Lipiodol" in small quantities is introduced through a small syringe with catheter attached, and the catheter is set in position under the fluoroscope; great care is necessary to avoid reflux into the lungs. The author can assess the possibility of successful operation from this procedure. Fistula between the œsophagus and trachea is common. Disorders in swallowing are considered; they usually occur in frail or premature babies with poor vitality. Vascular anomalies may produce compression of the œsophagus, a right-sided aorta or double aortic arch. Several congenital abnormalities are described and illustrated. Œsophageal spasm is not uncommon in early life, and short œsophagus and thoracic stomach are often associated with these conditions. Hypertrophic pyloric stenosis can usually be diagnosed clinically, but good information can be obtained by X-ray examination with an opaque meal. In a number of cases of stenosis, the author has seen no delay in emptying the stomach in spite of the presence of a large tumour. Diverticula of the stomach are not uncommon in infants, and ulceration is almost unknown in the stomach and duodenum. Duodenal obstructions are of congenital origin, and are frequently seen in mongols. In small intestine obstructions, a direct X-ray film and not an X-ray examination with an opaque meal should be relied on for diagnosis. Parasites, such as round worms, are frequently demonstrated. Intussusception rarely needs radiographic examination, but the enema is of value in its reduction. Megacolon is the commonest lesion met with in the lower part of the bowel. The author notes that the distal part of the colon is relatively large in infants. Non-obstructive and obstructive (Hirschsprung) lesions are described and well illustrated. Water intoxication may occur if large enemata are used. The book is very readable, and can be recommended to even the most experienced specialist.

An Atlas of Anatomy. By J. C. Boileau Grant, M.C., M.B., Ch.B., F.R.C.S. (Edin.); Fourth Edition; 1956. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 11¼" × 8¼", with 634 illustrations, many in colour. Price: £8 5s.

THE fourth edition of "An Atlas of Anatomy", by J. C. Boileau Grant, contains 80 new illustrations of the same high standard as the more familiar ones, with some excellent innovations in the form of basic diagrams of the limb and gut arteries and the bronchial tree distribution.

A most welcome addition is the triangle of the vertebral artery, and other nearby structures in the root of the neck are well covered.

The female perineum is dealt with in detail, and good figures of the foot joints and the shoulder capsule are added.

A wealth of detail (such as persisting median artery, *extensor digitorum brevis*, misplaced *palmaris longus*) is interspersed with variations of important structures such as the cystic artery. All in all the presentation is a most thorough and enjoyable one, both for undergraduate and for post-graduate student.

Low-Fat Cookery. By Evelyn S. Stead and Gloria K. Warren, with an introduction by Eugene A. Stead, junior, M.D., and James V. Warren, M.D.; 1956. New York, Toronto, London: McGraw-Hill Book Company, Incorporated. 8½" × 5½", pp. 200, with illustrations by Frank Sieminski. Price: $3.95.

THERE has been increasing interest over the past few years in diets of low fat content, and a cookery book giving dishes low in fat yet pleasant to eat would be very useful. E. S. Stead and G. K. Warren have produced a book called "Low-Fat Cookery" which purports to give such information. The authors are the wives of two professors of medicine in the United States of America. Their qualifications are that one is a trained dietitian, the other "has had practical experience of planning three meals a day for sixteen years, and of keeping happily fed a family who like to eat". The book contains about 150 recipes, from beverages to complex stews

and soups. With each recipe is given the fat content of a serving, but curiously often with the statement that this is materially reduced if the excess fat is removed before, during or after cooking.

Many of the recipes are complex, containing 10 or more ingredients—up to 15 in one case. A necessary piece of equipment is a good tin-opener, for most of the recipes have something out of a can. One, chicken and crab casserole, has 10 ingredients, five of which come from cans. While many of the dishes look very tasty, their complexity seems unnecessary.

Daily menus for a week are given to provide 25 and 50 grammes of fat per day, and they might have come from one of the most lavish of American women's magazines. Tables are given of the fat contents of a great variety of foods, many of which are unobtainable in Australia.

The book is an interesting one, and contains some choice recipes and a lot of useful information; but it would be of very little use in Australia for the purpose for which it is intended.

Essentials of Psychology. By Werner Wolff; Second revised and enlarged edition of "What is Psychology"; 1956. New York, London: Grune and Stratton, Incorporated. 8¾" x 5¾", pp. 400, with illustrations. Price: $6.50.

PROFESSOR WERNER WOLFF is to be congratulated on compressing a vast amount of data into his handbook which bears the caption "Essentials of Psychology". That more than 400 items are listed in the bibliography indicates the wide net of his search for material. He reminds us of a remark by William James that the world to an infant is "one big blooming buzzing confusion", and infers that a text-book of psychology can present a similar pattern to the student. There is the difficulty that living man moves in "a world of interpretations", and must be considered in a constantly changing environment. With this thought in mind, the author has endeavoured to put forward the broad principles which lie at the roots of behaviour. In the main he has achieved his purpose.

The book is divided into ten parts: "The Scope of Psychology", "The Physiology of Behaviour", "Perception", "Retention", "Thinking", "Intelligence", "Emotion", "Personality", "Attitudes", "Motivation".

The section on physiology is well illustrated, and shows us our dependence on nerve structure and the physiology underlying sensation. A section is devoted to the electrochemical basis of psychological activity, including the findings of electroencephalography.

The vagaries of perception, including imperceptions and illusions, are dealt with succinctly. The reader is reminded that all is not as it seems. Largely we see a world of our expectation. There is cited the experiment of showing cards on which is depicted a handsome Arab leaning over an ugly old woman; many of the subjects saw a beautiful girl.

The concept of *Gestalt* and the work of Pavlov, Freud, Adler and Jung receive adequate and sympathetic mention without bias.

Throughout the book reference is made to widely differing cultures in order to show what psychology must be considered in its individual frame. Thus memory depends on our dispositions. In our "book culture" we remember what we see, whereas illiterates such as the Chuckchee Indians memorize what they smell. The theme of the importance of the primitives is shown in a delightful love letter written by an Ojibwa belle; she shows in pictures the way to her wigwam. Such examples introduce us to the dreams and imaginations of children.

The section on intelligence deals with intelligence testing. As psychological testing is now a ten-million-dollar-a-year business in the United States of America, comment on its utility is indicated. The author admits the theoretical objections, which include the difficulty of testing emotional stability, but confirms its application.

Opportunely a chapter is devoted to the reactions to interference. Threats of interference tend to produce three main reactions—frustration, aggression or anxiety. Escape may show the way out, whether in withdrawal from responsibility or in the "keep smiling tactic" which conceals despair. The principles underlying the varied patterns of anxiety are described; but we are reminded of the importance of the details of social environment. "In an American village, for instance, social prestige has a great value; its loss produces loss of self-esteem and anxiety. In a metropolitan city, money has a great value; its loss may produce anxiety." Thus we see that values, the loss of which may produce anxiety, largely depend on conditioning by the environment or on factors of learning.

The physician will be interested in the pages on physiology and personality. The descriptions of masculine-femininty tests and the Kretschmer and Sheldon classification of body types are basic to an understanding of psychosomatic problems.

In an uncertain world Professor Wolff wisely gives no psychological panacea for international disharmonies. His experiments highlight the fundamental difficulties produced by differing racial cultures. The supreme values and overall life patterns in fundamentals are often basically as the poles apart.

"Essentials of Psychology" can be recommended as an introduction to psychology; but the reader is warned that in spite of its lucidity and illustrations, the condensation precludes profit by skimming. However, the discerning reader will appreciate the benefit of a factual and well-documented presentation.

Cytology of the Blood and Blood-Forming Organs. By Marcel Bessis; translated by Eric Ponder; 1956. New York and London: Grune and Stratton. 10" x 7", pp. 661, with illustrations. Price: $22.00.

THIS book is a very good translation by Dr. Eric Ponder of Dr. Marcel Bessis's *"Traité de cytologie sanguine"*. In his own words the author has set out "to reassess the classical ideas based on stained films by considering them in the light of recent techniques particularly those of phase microscopy, electron microscopy, cyto-chemistry, the use of the ultracentrifuge, the examination of blood cells by polarised light and by fluorescence microscope".

The book is divided into three parts. The first deals with technique. Descriptions of standard hæmatological procedures are followed by those describing recently developed techniques, such as phase-contrast and electron microscopy, cyto-chemistry, in-vitro culture *et cetera*. These techniques are clearly described and well illustrated. There is constant emphasis on underlying principles, and reasons for various steps are usually given so that the reader can, without difficulty, obtain a good understanding of the subject.

The second part deals rather briefly and very generally with the physiology and pathology of blood cells.

The third part comprises more than half the book, and is devoted to the systematic study of the various cell elements in the blood and of their precursors by techniques both old and new. This is a very comprehensive section which contains much useful and interesting material as well as much that is of academic interest only. Incidentally, most of the new techniques are not as yet of much practical value in elucidating routine diagnostic problems.

This book is impressively printed on high gloss paper. The reproductions of photomicrographs and diagrams are to be highly commended. The bibliography is most comprehensive. Whilst the book is highly recommended mainly for its contribution to academic hæmatology, it also presents a facet of the subject with which the present-day hæmatologist must be familiar.

Studies in Topectomy. Edited by Nolan D. C. Lewis, M.D., Carney Landis, Ph.D., D.Sc., and H. E. King, Ph.D.; 1956. New York and London: Grune and Stratton, Incorporated. 9" x 6", pp. 256, with illustrations. Price: $6.75.

THIS monograph is a collection of reports by a large number of investigators who have studied the effect of topectomy upon a group of 66 chronic schizophrenic patients. It forms only one aspect of a much larger research programme designed to evaluate psychosurgery as a therapeutic method, and in passing to accumulate basic information concerning the interrelation of frontal lobe structures and human behaviour.

The operation of topectomy is described. The 66 patients had been confined to mental institutions for at least three years, with the exception of a small number labelled pseudo-neurotic schizophrenics. All had failed to respond to other psychiatric methods. The patients were studied for three to six months pre-operatively and followed for three or four years post-operatively.

In the group of chronic deteriorated schizophrenics the results were not impressive; only 6·8% showed significant improvement, as judged by their suitability for release or their ease of management within the institution after operation. The best results were obtained in the pseudo-neurotic

schizophrenics, in whom the operation produced a diminution of "painful affect associated with mental conflict".

No permanent reduction in intelligence was produced by the operation, and no additional psychiatric abnormality was added to the existing mental state of any patients.

Many references are given to works on other aspects of psychosurgery, and from one of these a list of the disorders which are all alleviated by topectomy is quoted. It is likely that the majority of the patients in the present series would have been better treated by prefrontal lobotomy.

This is a work of interest only to those practising psychosurgery or psychiatry.

Pelvimetry. By Herbert Thoms, M.D.; 1956. New York: Paul B. Hoeber, Incorporated. 9¼" × 6¼", pp. 120, with illustrations. Price: $5.00.

HERBERT THOMS is an obstetrician and gynæcologist of world standing. He has contributed many papers to the literature on X-ray studies of the female pelvis, and this small book covers clearly and concisely the whole field of pelvic conformity in relation to child-bearing. It is a book for the obstetric specialist, and should be read by all studying for that field. It is a book more for the obstetrician than for the radiologist.

The author states clearly that the purpose of X-ray pelvimetry studies should be to assess the pelvic factor only—not to decide whether vaginal or Cæsarean delivery should be carried out. It is generally accepted now that clinical methods fall far short of requirements in studying the shape and capacity in the upper part of the pelvis; X-ray examination alone gives us this, sometimes essential, information. There is a good chapter on the clinical significance of pelvic variations. In it the following statement is made: "When abnormal pelvic conformation is present, all important pelvic levels must be critically assessed for capacity if the pelvic assessment is to have real prognostic value." There is an excellent chapter on mid-plane contraction, and the section on "Rarer Forms of Pelvic Abnormality" is interesting. The discussion on the development of the pelvis and the influence of nutrition on its conformation is stimulating reading.

This is an excellent small book, largely on original work and therefore written with authority. The type and illustrations are good. It should be read by every serious obstetrician, who will find his interest stimulated afresh in the variations of the female pelvis, which we often ignore, while we hope for the best in labour.

Untersuchung und Beurteilung des Herzkranken. By H. W. Knipping, W. Bolt, H. Valentin and H. Venrath; 1955. Stuttgart, Germany: Ferdinand Enke. 9½" × 6½", pp. 461, with illustrations.

PROBABLY the most well-informed cardiac clinic in Western Germany is the Medical University Clinic at Cologne. Professor H. W. Knipping and his assistants, Dr. W. Bolt, Dr. H. Valentin and Dr. H. Venrath, have compiled an account of their beliefs and practice under the title (translated) "Investigation and Review of Heart Disease". This is the most important and valuable statement to emerge from post-war Germany, a country which contributed so much to the investigation and treatment of heart and renal disease in the early part of this century. Before World War II, Germany was in the forefront of cardio-thoracic surgery, and this book is dedicated to the pioneer Ludolph Brauer. In a philosophical introduction, the authors plead for a wider recognition of their famous predecessors including Forssmann, the first to pass a catheter into his own heart. They also display an understanding of the patient's point of view, and a recognition of the discomfort he may be caused by modern forms of investigation. They point out the necessity for specialization within the fields of internal medicine and the rapid progress made by such specialized teams in Great Britain and the United States of America.

The production is divided into sections on the following subjects: (*a*) investigation of heart disease, (*b*) pre-operative diagnosis of heart disease, (*c*) functional studies in relation to prophylaxis of heart disease and in athletics, (*d*) *cor pulmonale*. This is an odd assortment of subjects, but the reader will find a good description of the diagnosis of congenital heart disease, of the elements of modern electrocardiography and, of course, of the application and results of the authors' own researches in their closed-system spiroergometer. There are also excellent descriptions of regional angiocardiography, the use of isotopes in localizing abnormal circulatory flows, cardiac volume *et cetera* and the diagnosis of *cor pulmonale*. There is a good section on anæsthesia and pre-operative preparation of patients for cardiac surgery and on the management of cardiac arrest. The chapter on radiology includes a brief description of all modern techniques and many obsolete ones such as orthodiagraphy. The reproduction of radiographs is particularly good. Some of the tables are overloaded, and the text follows the rather discursive path familiar to readers of Continental medical literature. Great pains have been taken to acknowledge previous workers in each field, and an impressive bibliography concludes the book. The breadth and logic of the presentation must be respected, and the result is an original, practical survey of the subjects chosen.

A Text-Book of Surgical Pathology. By Charles F. W. Illingworth, C.B.E., M.D., Ch.M., F.R.C.S.(Ed.), and Bruce M. Dick, M.B., F.R.C.S.(Ed.); Seventh Edition; 1956. London: J. and A. Churchill, Limited. 9½" × 6¼", pp. 738, with 322 illustrations. Price: 63s.

IT is seven years since a new edition of Illingworth and Dick's "Text-Book of Surgical Pathology" appeared. We have now the seventh edition. Much which is commendable has been achieved. Despite increase in knowledge of basic surgical problems, this book is, if anything, slightly smaller than its immediate predecessor. This has been made possible by the elimination of descriptions of diseases which are now cured by modern therapeutic measures, and which therefore contribute no pathological material for examination. On the other hand, new sections have been written, as befits a standard text-book written "for graduates and senior students".

Some of these new sections we read with a feeling of disappointment. For example, the subject of shock is discussed in the light of modern views on fluid and electrolyte balance; but the subject matter, while clear and concise, is mainly clinical, while the effect of various disturbances on body cells—surely the concern of the pathologist—is given scant attention.

It would be unusual if such a book contained only statements which were of universal acceptance. For example, it is stated that cancer of the thyroid gland is specially prone to develop in glands already altered by disease. This is perhaps true of the malignant adenoma; but the other forms, as is stated in this book, are more than twice as common in men as in women and may readily arise in previously normal glands.

We could go on differing on individual points. This does not detract from the obvious attraction and value of this famous text-book.

Pharmacognosy. By Gathercoal and Wirth. Third Edition, thoroughly revised by Edward P. Claus, Ph.D.; 1956. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 10¾" × 7¾", pp. 731, with 306 illustrations and one plate in colour. Price: £6 17s. 6d.

PHARMACOGNOSY is a subject which has undergone a vast change of approach in the last few years. It is no longer primarily a study in the morphology of plants and animals from which drugs are obtained—a somewhat dry approach. Now students of this subject are becoming increasingly interested in the constituents and the factors which govern the formation of drugs in the plants and animals. This change can largely be attributed to the rise of the antibiotics. Yet again the pharmacognosist pursues the subject with the drug constituents as the primary feature in the classification instead of the older natural system.

This well-produced, outstanding book, in the third edition, brings this new approach before the student. In the first part of the book few changes have been made, as this section deals with the older plant drugs and is presented in a monograph form. However, these have now been arranged according to their constituents and are classified as alkaloids, glycosides, volatile oils, fats and waxes and so on.

The new edition is notable for the addition of four new chapters dealing with antibiotics, immunizing biochemicals, allergens and allergenic preparations and pesticides. The antibiotics and immuno products are discussed in individual monographs; but the two remaining subjects are dealt with in a more general manner. An indication of the comprehensive nature of the book is the inclusion of nystatin and polymyxin and a description of the production of poliomyelitis vaccine. It is often forgotten that pharmacognosy includes the endocrine products, and these are dealt with very fully in a well-planned chapter in which these drugs are discussed from the practical standpoint associated with their manufacture.

The book is well illustrated with excellent photographs, and the standard of printing and production represents the best of current American practice.

This is a book for the chemist in industry, for the hospital pharmacist, and for those members of the medical profession whose interest goes beyond the daily problems of therapeutics.

Practical Hæmatology. By J. V. Dacie, M.D. (Lond.), M.R.C.P. (Lond.); Second Edition, 1956. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 234, with 43 illustrations. Price: 20s.

A SECOND EDITION of "Practical Hæmatology" by J. V. Dacie, Reader in Hæmatology in the Postgraduate Medical School of London, will be welcomed by all who are interested in this subject. Dr. Dacie is, of course, well known for his work on the hæmolytic anæmias. The new edition is an excellent book, clear, efficient and unpretentious. In his preface the author states that he has revised and rewritten his work in an attempt to make it an up-to-date and practical laboratory guide. The revision has been very thorough: the first edition was well and clearly written; the new one has been meticulously revised, passage by passage, pruned or amplified according to need, and the result is a model of lucidity. Very few medical books are so utterly free from padding. So much for the manner of the book; as to the matter, it is a practical laboratory guide, not a text-book on hæmatology or a treatise on the disorders of the blood. It does not attempt to compete with the larger standard works on the subject, but is a most useful, indeed an indispensable, supplement to them. It is intended for hospital pathologists, post-graduate students of clinical pathology and senior laboratory technicians in their day-to-day work. Such workers must have the new edition, not only because of its merits, but because the first editions of Dr. Dacie's book, wherever one sees them, are well worn by constant use, the best tribute a book can have. He has not attempted to describe the appearances of normal or pathological blood cells when stained by Romanowsky dyes. As he puts it: "To do this adequately would have increased the size and price of the book beyond what was intended." However, brief descriptions of blood cells which have been stained supravitally have been retained, and illustrations of Heinz bodies and siderotic granules have been added. Since the appearance of the first edition in 1950, many important advances in the laboratory investigation of blood diseases have been made, especially with regard to the hæmorrhagic disorders, and these are well and competently dealt with. A chapter on the choice of hæmatological tests in various clinical conditions should be read by every medical practitioner. The author condemns unnecessary and indiscriminate use of the laboratory; at the same time he insists on a high standard of technique, and will spare no pains to perform detailed studies and to obtain as precise a result as possible when this is necessary. The inherent errors of the red cell count are described at some length. Dacie's opinion that "the value of the red cell count in diagnosis is seldom great" is well known. Two types of case in which an accurate red cell count may be important are polycythæmia and "mild megaloblastic anæmias". Dacie writes also that "quantitative counts and 'absolute values' rarely give diagnostic information that cannot be appreciated from the inspection, by a trained observer, of a well-spread and well-stained blood film". The idea behind this book is to lay aside every weight (of convention and tradition in blood counting) and the sin (of failing to think) which so easily besets the routine laboratory worker and the busy physician, and to illuminate and perfect the more difficult aspects of the examination of the blood.

Internal Medicine: A Physiologic and Clinical Approach to Disease. By Robert P. McCombs, B.S., M.D., F.A.C.P.; 1956. Chicago: The Year Book Publishers, Incorporated. 9" × 6", pp. 727, with illustrations. Price: $10.00.

THIS book contains 49 illustrations. These are mainly X-ray plates of indifferent quality, and while there are some useful diagrams which outline the physiological mechanisms involved in certain clinical conditions, some of the diagrams are confusing.

The volume claims to be a summary of the most important clinical facts, physiological concepts, diagnostic methods and therapeutic measures of use in the study and management of internal diseases. On the subjects included and within the intended scope, the book is reasonably informative; its value is enhanced by a short bibliography at the end of each chapter. The author unfortunately regards diseases of the nervous system as an independent specialty, and there is no section dealing with nervous diseases.

The work cannot be recommended as a well-balanced graduate or undergraduate general medical text-book.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"An Atlas of Anatomy", by J. C. Boileau Grant, M.C., M.B., Ch.B., F.R.C.S. (Edinburgh); Fourth Edition; 1956. London: Baillière, Tindall and Cox, Limited. 11" × 8½", pp. 562, with 714 illustrations, many in colour. Price: 120s.

To the last edition, reviewed in this journal in March, 1953, eighty new illustrations have been added.

"Handbook of Biological Data", edited by William S. Spector; October, 1956. WADC Technical Report 56-273, ASTIA Document No. AD110501. Prepared under the direction of the Committee on the Handbook of Biological Data, Division of Biology and Agriculture, The National Academy of Sciences, The National Research Council. Wright Air Development Center, Air Research and Development Command, United States Air Force, Wright-Patterson Air Force Base, Ohio. 10¾" × 8½", pp. 620.

Contains tabular data and graphs, charts and diagrams in the broad areas of plant, animal and pre-clinical medical sciences.

"The Young Handicapped Child: Educational Guidance for the Young Blind, Cerebral Palsied and Deaf Child", by Agatha H. Bowley, Ph.D., F.B.Psy.S., with a section on "The Young Deaf Child", by L. Gardner, B.Sc., Dip.Ed., Dip.Psych.; 1957. Edinburgh and London: E. and S. Livingstone, Limited. 7¾" × 4¾", pp. 136, with 36 illustrations. Price: 10s. 6d.

Written for the ordinary parent and teacher.

"Principles of Epidemiology", by Ian Taylor, M.D., M.R.C.P., D.P.H., and John Knowelden, M.D., D.P.H.; 1957. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 308, with 26 illustrations. Price: 30s.

For students and practitioners.

"Kinetics and Thermodynamics in Biochemistry", by H. Geoffrey Bray, D.Sc. (Birm.), and Kenneth White, B.Sc. (Lond.), Ph.D. (Birm.), F.R.I.C.; 1957. London: J. and A. Churchill, Limited. 9½" × 6", pp. 356, with 71 illustrations. Price: 42s.

Designed primarily for the honours student in biochemistry and the post-graduate worker entering biochemistry; also for biochemists experienced in fields in which thermodynamics or kinetics have not so far been applied, and for physical chemists.

"Injuries of the Hand", by Ronald Furlong, F.R.C.S.; 1957. London: J. and A. Churchill, Limited. 9¾" × 6¼", pp. 226, with 99 illustrations. Price: 36s.

Describes the current teaching and practice of the Orthopædic Department of St. Thomas's Hospital, London.

"A Synopsis of Regional Anatomy", by T. B. Johnston, C.B.E., M.D.; Eighth Edition; 1957. London: J. and A. Churchill, Limited. 8" × 5¼", pp. 460, with 20 plates and 19 text figures. Price: 28s.

Revised but not drastically altered from previous editions.

"Hale-White's Materia Medica, Pharmacology and Therapeutics", by A. H. Douthwaite, M.D., F.R.C.P. (Lond.); Thirtieth Edition; 1957. London: J. and A. Churchill, Limited. Price: 24s.

The previous edition was published in 1952, the first edition in 1892.

"The Child and the Outside World: Studies in Developing Relationships", edited by Janet Hardenberg, M.B.; 1957. London: Tavistock Publications, Limited. 8" × 5¼", pp. 202. Price: 16s.

Writings concerned chiefly with the older child addressed to teachers and case workers.

"A Therapeutic Index", by C. M. Miller, M.D. (Lond.), M.R.C.P. (Lond.), and B. K. Ellenbogen, M.D. (Liverpool), M.R.C.P. (Lond.); Second Edition; 1957. London: Baillière, Tindall and Cox. 7¼" × 4¾", pp. 168. Price: 12s. 6d.

Considerably revised and expanded since the publication of the first edition in 1955.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

ECOLOGY AND DISEASE.

WITHIN recent years there has been a great number of papers written about degenerative diseases in man; and more particularly concerning atherosclerosis and coronary artery disease. Whereas at one time it was thought that these diseases were merely the accompaniment of an increasing expectation of life, it is now thought by most medical people that these diseases are more properly an accompaniment of our mode of living. A majority of medical opinion has been interested in the epidemiology of atherosclerosis, and has connected the eating of fat with the occurrence of atheroma, either speculatively or definitively. This speculation grew from the experiments performed by Ignatowski in 1908. These were the now classical experiments of repeatedly injecting rabbits with cholesterol, and eventually producing generalized atherosclerosis. Since that time the work has spread, until now many papers are available, most of which point to a relationship between fat in the diet and atheroma. Recent work has extended this supposition to the consideration of fatty acids both saturated and unsaturated in this field. The whole subject has received many airings in the editorial columns of this journal,[1] and whilst the recent papers on fatty acids are both pointed and in need of review, it is probably important to dwell upon ecology both past and present before discussing any *ad hoc* implications.

An ecological study is of value for several reasons: firstly from the viewpoint of perspective alone; secondly because of the direct relationship to the emergence of coronary heart disease as a problem of major importance in man; thirdly because an evolutionist may have a relevant comment to make on this problem; fourthly because epidemiology applied to such an insidious and gradual process as coronary artery disease is a departure of recent years, and one of great importance. Animals in their natural surroundings choose diets which for them are nutritionally adequate; and it is said that man, in a wide variety of environments, does the same, and therefore demonstrates a food instinct. However this may seem, it is difficult of proof, and often seems completely wrong, as the choice of food is made at many quite different levels. Man's choice of food depends on many unrelated considerations, as shown by J. Yudkin.[1] Our own choice of food is redolent of our eclecticism, and in these days is even transepicurean, in that it is most often related to a brand of a particular food. Other peoples and more primitive man are governed by more mundane principles. Limitation by geography and distance is perhaps the most potent. Other considerations are religion, prejudice, weather, storage, transport and food technology. Most people living in primitive conditions now and in the past lead a more or less precarious existence as regards exposure and food supplies. Under these conditions of life only the physically well equipped will survive.

The early Egyptians were great agriculturalists;[2] and it is a popular belief, no doubt owing to the site of the river Nile and its delta, that agriculture as a science was born there. Most Egyptians ate spelt, bearded wheat or barley made into bread. Most of the vegetables familiar to us were eaten, as well as fruits such as grapes, figs and dum palm. A lot of fish was netted in the Nile, but meat eating was confined to the nobility. It should be recalled that atheromatous mummies have been exhumed. In the *tundra* areas the diet usually consists of meat and fish, as it has done for centuries. There is usually no exposed soil, and there is little edible vegetation beyond lichens. When the weather permits, the inhabitants of these regions are perpetually on the search for food, and many die of starvation; this fate, for example, is common amongst Eskimos. The warm East eats now, as it always has done, a great deal of rice, a lesser amount of soy bean, and occasionally pork or fish. Other foods are seldom eaten save by a privileged few (especially is isolated areas). The people who inhabit tropical rain forest are multifarious, but the staple item of diet is one or other root vegetable providing carbohydrate. Desert peoples are carbohydrate eaters, but they usually manage to supplement this with cheese from their own animals. All these tribes and peoples, together with others, as, for instance, the people of Java, Borneo and Tristan da Cunha, live off local agriculture without moving far afield. As they have virtually no income, they import no food. There is a notable paucity of protein and fat.

Two hundred years ago the struggle for existence and the diet in Western Europe were similar in type to the ones mentioned above. The diet in England was itself divided geographically; in the south bread and cheese, with a little meat and bacon, were eaten, while in the north the diet was oatmeal and milk. Only large ports like London had access to imported foods. How then did the changes in our diet arise? The Industrial Revolution in Europe, and more particularly in England, brought about a great numerical and technical expansion. The numerical increase created a demand for food which local agronomy could not satisfy. With the forced importation of food came food storage and processing. The Industrial

[1] M. J. AUSTRALIA, August 26, 1944; July 26, 1952; March 14, 1944; April 4, 1953; December 3, 1955; June 2, 1956; September 22, 1956.

[1] *Lancet*, May 12, 1956.
[2] "The Newer Knowledge of Nutrition", 5th Edition, 581.

Revolution made coal abundant and cheap as a fuel, and this enabled food to be cooked and made palatable in ways which, previously, had been impossible. It is from this time that our modern diet stems. Americans, Scandinavians and the Europeans of the Commonwealth today take about 40% of their total Calories as fat. These countries are the ones in which atheroma is a problem. There is a striking correlation between amount of fat eaten and incidence of atheroma, as A. Keys[1,2] and others have shown. R. B. Bronte-Stewart *et alii*[3] have suggested that an important factor is the amount of unsaturated fatty acid present in the diet. They have shown that unsaturated fatty acids will reduce the amount of cholesterol and β-lipoprotein present in the blood of a person on a normal European diet. H. M. Sinclair[4] has carried this supposition further. He wonders how far processing of food has affected our health. We may well wonder how man's ecology is affecting his health. Keys is convinced that we need to change our diet and eat less fat. Certainly, there is nothing revolutionary in the idea, as it would be a reversion towards our former state. Whether or not man would accomplish such a change is another matter. As the history of life is one of ebb and flow, an evolutionist would have a great interest in man's reaction. It is by no means unknown for an organism to develop in an unfortunate direction to the point of extinction. As medicine becomes more complex, we narrow our field of vision to that in which we work. For that reason perhaps we should lift at least some of our investigations of man's ills into a broader realm. When we have done so, we may find that our opinions are increasingly macabre.

Current Comment.

TUBERCULOSIS MORTALITY IN EUROPE.

A STEADY DECREASE is occurring in the number of deaths from tuberculosis in Europe. This has been going on for some time, but figures released recently by the World Health Organization show a striking drop in the death rate from tuberculosis during the period from 1950 to 1955 in most countries of Europe. For example, the death rate per 100,000 of population dropped from 13·8 (in 1950) to 6·3 (in 1955) in Denmark, from 58·1 to 31·3 in France, from 143·6 to 63·0 in Portugal, from 36·4 to 14·6 in England and Wales, and from 53·6 to 19·1 in Scotland. Another interesting aspect of the trend is the decrease in mortality from tuberculosis in relation to total mortality. In Denmark, which has the lowest figures for Europe, in 1950 tuberculosis accounted for 1·5% of deaths from all causes; in 1954 the proportion had dropped to 0·9%. In Portugal, which has the highest figures, progress was even more striking, the proportion dropping from 11·7% in 1950 to 5·6% in 1954. The figure in England and Wales dropped from 3·1% to 1·6%, and in Scotland from 4% to 1·8%.

Tuberculosis of the respiratory system, and in particular pulmonary tuberculosis, remains the leading cause of death among the various forms of tuberculosis. In 1950 the proportion of deaths from tuberculosis of the respiratory system to total tuberculosis mortality ranged from 72·4% in Iceland to 89·1% in West Berlin; the figure for England and Wales was 88·2% and for Scotland 87·9%. In 1954 mortality from respiratory tuberculosis represented an even larger share of the total tuberculosis mortality in all countries except Spain, Northern Ireland, Portugal and Switzerland, where the figures were little if at all altered; the figure had risen to 100% in Iceland, to 93·4% in West Berlin, to 89·5% in England and Wales, and to 89·5% in Scotland. The report further shows that more men than women die from this form of the disease; the proportion is more than two to one in England and Wales. There has also been a considerable change in the age distribution of deaths from tuberculosis of the respiratory system; before World War II the majority of victims were women between the ages of twenty and thirty years, and men between the ages of forty and fifty-five years, but nowadays deaths are most numerous among people past sixty years of age, men and women alike.

The World Health Organization report stresses the fact that despite the encouraging progress noted, tuberculosis remains one of the ten major causes of death in Europe. It is still considered as a reliable index of a country's development, not only in public health, but also in the social and economic spheres. Justifiably, governments continue to put control of tuberculosis among the chief items in their health programmes, and still consider it as one of the most important social diseases.

TREATMENT OF HÆMOPHILIACS WITH HUMAN ANTIHÆMOPHILIC FACTOR.

THE treatment of hæmophilia, or more particularly the control of hæmorrhage in hæmophiliacs, presents formidable problems. In 1954 R. G. Macfarlane, R. Biggs and E. Bidwell[1] reported the preparation of antihæmophilic factor in very high potency from animal blood. This opened up new possibilities, but it is only too obvious that complications are likely to be associated with the use of an animal product of this kind in the treatment of human beings, especially when the treatment is recurrent. The logical answer is a potent preparation of human antihæmophilic factor, but this has presented great practical difficulties. Now R. A. Kekwick and P. Wolf[2] have made substantial progress in overcoming these difficulties and report their experience with a concentrate of human antihæmophilic factor, which is stated to contain not less than 85% of the activity of the material from which it was initially prepared. They describe their method of preparation and go on to report clinical experience of its use in six cases of hæmophilia. The particular situations in which it was used included single and multiple tooth extractions, hæmorrhages into the stomach and joints, and radical surgical operation on the buttock and rectum. The results were gratifying, with arrest of internal and external hæmorrhage and prevention of abnormal bleeding during surgical intervention. This means not only control of the dangers directly associated with loss of blood, but avoidance of the well-known complications of hæmophilia such as permanent joint lesions, and of the unhappy situation in which the surgeon is loath to undertake essential surgical proceedings in the hæmophiliac.

In the past some measure of control has been exercised by the transfusion of fresh blood, fresh plasma or stored frozen plasma; but this has been attended by the risk of overloading the circulation because of the low antihæmophilic potency of human blood or plasma and the consequently large amounts that need to be transfused. For this reason the provision of a concentrate of which 100 millilitres are equivalent in activity to one litre of fresh plasma, and which can be made available in a few minutes by the reconstitution of stable freeze-dried material, is, as Kekwick and Wolf state, a substantial advance in the treatment of hæmophilia. They hope to achieve a further tenfold purification, so that the activity of one litre of fresh plasma could be injected in a volume of 10 millilitres. To do this without substantial loss in the total activity of the starting material is, they state, at present difficult. Meanwhile the potency of the present

[1] "Cardiovascular Epidemiology", 50ff, 135ff, 175ff.
[2] *Arch. Int. Med.*, March, 1954.
[3] *Lancet*, April 28, 1956.
[4] *Lancet*, April 7, 1956.

[1] *Lancet*, June 26, 1954.
[2] *Lancet*, March 30, 1957.

preparation is high enough to obviate the dangers of circulatory overloading during extensive treatment.

Kekwick and Wolf point out that the antihæmophilic activity of some animal plasmas is considerably greater than that of human plasma, but the use of antihæmophilic preparations from animal sources is seriously restricted by the hazard of allergic reactions following the repeated administration of heterologous proteins. This is not the case with material from human plasma. Commenting on reports of hæmophiliacs becoming resistant to therapy after repeated treatment over long periods even with human plasma and products derived from it, Kekwick and Wolf draw attention to one of their cases, in which repeated infusions of their preparation were given over a period of five months; no resistance to therapy appeared, and laboratory tests at the end of the period showed no evidence of the development of even traces of a specific circulating anticoagulant.

Even with the advantages accruing from the use of such a preparation as that produced by Kekwick and Wolf, the problem of the management of hæmophiliacs is still considerable. They put it this way. If we assume on the basis of their limited clinical experience that a single crisis in a hæmophiliac might on the average be countered by the injection of an amount of antihæmophilic factor equivalent to that in two litres of fresh human factor, this would represent the plasma derived from about seven blood donations. The total number of hæmophiliacs in England is probably close to 2000; so to provide each with a single injection each year would require 14,000 blood donations—about 2% of the current total annual donations. The processing of the plasma from 14,000 blood donations by the procedure found necessary to ensure a maximum recovery of antihæmophilic factor presents a formidable problem, the solution of which is not yet clear. It is, of course, of the utmost importance that the greatest possible yield be obtained also of the other constituents of the blood in the fractionation process. Apart from anything else, the mere collection of the blood required to meet the need of the hæmophiliac population would be a major administrative task.

DIAGNOSIS AND TYPING IN LEPTOSPIROSIS.

LEPTOSPIROSIS, which has long been of interest in Queensland, is in fact a disease of world-wide distribution. Coming within the category of the zoonoses, it is transmitted to man primarily through the urine of infected animals contaminating water and sometimes food, and also by direct contact with infected animals. Of the causal organisms, the leptospires, a large number of different types exist, and it is possible to distinguish between them only through laboratory tests involving serological reactions. However, owing to the great diversity of methods and techniques, considerable confusion arises, which makes comparison of results obtained in laboratories in different parts of the world exceedingly difficult. The need for some order in this confusion is obvious, and in order to lay the foundations for a standardization of techniques used in the diagnosis of leptospirosis and the typing of pathogenic strains of leptospires, the World Health Organization convened a study group, which met in November, 1955. Its report has now been published.[1]

The report stresses the fact that agglutinogens, determined by agglutination and cross-absorption reactions with immune rabbit serum, remain the best criteria for the classification of the leptospires into serotypes. Standardization of sera—that is, of specific reference sera to be made available to research workers—is one of the fundamental requirements for standardization techniques. The members of the study group examined the results of comparative assays of sera corresponding to six serotypes of leptospires carried out by eight laboratories in different countries. The conclusion reached was that reference sera should be prepared, as far as possible, for all the types and subtypes at present well established, and that they should be made available in the form of freeze-dried preparations. Reference laboratories, set up in various parts of the world, in collaboration with the Food and Agricultural Organization, would then receive the sera and hold them in readiness for supply to national laboratories and specialist research workers. The laboratories would also provide cultures of leptospiral strains and would assist workers wishing to identify and classify new strains. An additional duty of the reference laboratories would be to train specialist laboratory technicians.

As a result of the recommendations made by the study group, reference centres for leptospirosis have already been designated at Amsterdam, London, Tokyo and Washington, D.C. Additional centres are expected to be designated in the near future. The report anticipated that early in this year standard reference sera in dried form would have been prepared for the main serotypes of Leptospira.

With regard to the problem of diagnosis, the report describes the methods which the members of the study group deem particularly suitable for this purpose—namely, blood culture and animal inoculation in the initial stage of the disease, and during the second and third weeks the agglutination-lysis test, straight agglutination (microscopic) and the complement-fixation test. The last-mentioned of these is of restricted application at present and is not recommended for use by routine diagnostic laboratories.

[1] "Diagnosis and Typing in Leptospirosis: Report of a Study Group", World Health Organization Technical Report Series No. 113; December, 1956. Geneva: World Health Organization. 9¼" × 6¼", pp. 11. Price: 1s. 9d.

HEART-LUNG PREPARATIONS.

THE widening scope of intrathoracic surgery has promoted further research into heart-lung preparations with the intention of devising techniques which will allow a dry operating field without general circulatory arrest in the patient. W. J. Potts *et alii*[1] have shown that respiration can be maintained adequately in the dog by diverting some of the blood through homologous lungs. W. T. Mustard and A. L. Shute[2] have described experimental intracardiac surgery in which either a whole lung, or a lobe of lung, removed from one dog, has been used to oxygenate the blood of another dog. Wally Gordon[3] has been experimenting with the use of isolated dog lungs as an extracorporeal oxygenator. The donor dog was anæsthetized and exsanguinated. By means of a bilateral thoracotomy, involving the sternum, the thoracic viscera were exposed. The azygos vein, *venæ cavæ* and ascending aorta were divided between ligatures. The trachea was severed, and the heart and lungs were removed *in toto*. Intermittent positive pressure ventilation was maintained by means of an endotracheal tube. The lungs were then washed out; it was found that 10% "Dextran" solution was the most suitable fluid for this purpose, because "Dextran" caused no pulmonary œdema, whereas most other fluids did. Any lung after two hours' exposure, however, developed pulmonary œdema on perfusion. The recipient dog was anæsthetized, and heparin was injected intravenously. Cannulæ were inserted into the azygos vein, *venæ cavæ* and femoral, carotid or subclavian artery. The animals were connected by tubing in a circuit containing a rotary pump, a filter and a venous reservoir filled with the blood collected in exsanguinating the donor dog.

In two experiments blood was withdrawn from the inferior *vena cava* of the recipient dog, circulated through the homologous isolated lungs, and returned to the recipient via the superior *vena cava*. Both recipient dogs made uneventful recoveries, and these two experiments would suggest that the simple passage of blood through isolated homologous lungs is not harmful to the dog. Studies on the blood oxygen content in these experiments indicate that efficient oxygenation is accomplished by the isolated lungs for at least thirty minutes.

[1] *Surgery*, January, 1952.
[2] *Surgery*, October, 1951.
[3] *Guy's Hosp. Rep.*, January, 1957.

The remaining data available from these experiments reveal the shortcomings of the technique at present used. All the 20 dogs which underwent the experiments, as recipients with arterial replacement, were alive at the end of the operation and breathing naturally. However, 17 never recovered consciousness and died within twelve hours. Only three dogs recovered sufficiently to drink and walk, and two of these died within forty-eight hours. The one dog which survived made an uneventful recovery, having had a complete by-pass for fifteen minutes. Post-mortem examination of 12 of the dogs revealed pleural cavities filled with fluid blood. The amount of blood was sufficient to cause death from hæmorrhage and pulmonary collapse. This cause of death cannot, however, explain the failure of the dogs to recover consciousness after operation, as all the animals had a pleural drain inserted at the end of the operation. The drains remained in place until the dogs were placed in their cages, and up to that time there was little or no drainage.

However disappointing the experiments may seem at first sight, the goal of a satisfactory oxygenating by-pass for use in human surgery is worthy and seems possible. Gordon has pumped citrated human blood through his preparation for thirty minutes with this goal in mind. This manœuvre was accomplished successfully.

ATTENUATED LIVE-VIRUS POLIOMYELITIS VACCINE.

THE name of Albert B. Sabin, of Cincinnati, is associated in most people's minds with the idea of attenuated live-virus poliomyelitis vaccine. Work aimed at the production of a satisfactory vaccine of this kind has occupied Sabin for some years, and he has recently[1] assessed its present status. He points out that the obvious reason for trying to learn all we can about immunization with an orally given, living attenuated poliomyelitis virus vaccine is to see whether it may be possible to reproduce the long-lasting immunity conferred by natural infection without the varying associated risk of paralysis. There are also the secondary considerations of the advantages of a vaccine that can be given by mouth instead of by injection, and of the possibility that widespread oral use of highly attenuated poliomyelitis viruses may eliminate the naturally occurring virulent strains, just as the smallpox virus was eliminated from many parts of the world by the use of the living vaccine against smallpox. Sabin summarizes various studies which, it would seem, have established beyond doubt that immunization of human beings by the oral route not only is possible but has been successfully accomplished. Since attenuated strains of poliomyelitis virus were found to vary greatly in the extent of their residual neurotropism for the most sensitive lower motor neurons, as well as in the homogeneity of their populations, the crucial problem was to find strains that were so highly attenuated and homogeneous that one would be justified in using them in increasingly larger numbers of human subjects in those stepwise tests that must precede any trial of such a vaccine on a large scale. Sabin describes the finding of such strains after tests on the progeny of large numbers of individual virus particles. He sums up the present position with the comment that it may be said that much has been learned about the basic principles underlying immunization with attenuated poliomyelitis viruses given by the oral route, and that we now have strains derived from single particles of virus that are sufficiently highly attenuated and stable under appropriate conditions of cultivation to justify their use for the next stepwise studies on immunization of human beings. Remarking that experience has taught him the importance of accentuating the negative, Sabin stresses the fact that the time is not yet ripe for so-called mass trials of an orally given vaccine, but only for those tests on increasingly larger groups that must precede any consideration of tests on a large scale. In view of the rivalry that many people envisage between the schools of thought represented respectively by Sabin and by Salk, Sabin's comment on the Salk vaccine is interesting and important: "I also want to accentuate the positive by saying that the Salk vaccine is the only poliomyelitis vaccine available for public use at this time and that advantage should be taken of its protective effects to the maximum extent of its availability." Sabin adds that some of his earlier reservations disappeared with the demonstration that the antibodies and immunity produced by the Salk vaccine did not interfere with the alimentary infection produced by an orally given, attenuated virus or naturally acquired virus. This is the position as it stands. It is obvious, as Sabin states, that we still have a great deal to learn before the ultimate goal of complete elimination of poliomyelitis is achieved; but we have gone a long way further than many people dared to hope a few years ago.

[1] *J.A.M.A.*, December 29, 1956.

CONGENITAL HEART DISEASE.

IT is not common to have more than one child in a family with congenital heart disease, although it is said to occur more frequently than would be expected by chance. M. Campbell and D. MacCarthy[1] have reported a series of families with *situs inversus* and congenital heart disease. Their "major" case report is of a family in which two cousins had *situs inversus* and congenital heart disease. Their mothers were sisters; and in addition one of the cousins had parents who were also cousins. Campbell and MacCarthy report details of three other patients with *situs inversus* and congenital heart disease, who have one or more near relatives with *situs inversus*, and often some other form of congenital heart disease. This is an interesting paper in view of the suggestion that dextrocardia is inherited as a Mendelian recessive character. The malady would therefore be more common in the children of cousin marriages. As the suggestion is by no means universally accepted, further evidence for or against it would be welcome.

RADIOACTIVE IODINE AND BENIGN THYROID DISEASE.

THE possible dangers arising from the treatment of benign thyroid disease with radioactive iodine have been discussed by Alton Ochsner.[2] He points out that unquestionably hyperplasia of the thyroid gland can be readily controlled by the use of radioactive iodine as effectively as by surgical methods. However, an obvious disadvantage of the use of radioactive iodine is that it is not easy to determine the exact amount required; hence it is possible to produce too much destruction of the thyroid gland, with resultant hypothyroidism. Perhaps this will not be regarded as a serious complication, because substitution therapy is relatively easy, but there are much greater dangers. Ochsner points out that an agent such as I^{131} may produce changes which subsequently lead to neoplasms. He cites evidence from the literature to show that children who have been treated by irradiation of thymic enlargements in childhood and infancy have a greater tendency to the development of leuchæmia and carcinoma of the thyroid, as well as other forms of cancer, than those who have not been irradiated. He states that the findings are particularly significant because the irradiation dosages used were small; the statistics indicate that irradiation in relatively small doses (from 200*r* to 600*r*) is dangerous as regards the subsequent development of malignant lesions.

Oschner points out that it is only recently that the danger from irradiation of the thymus has been appreciated; this is understandable, because it takes some years for irradiation damage to become evident. However, it is likely that in the future many patients who have been given radioactive iodine therapeutically may develop

[1] *Guy's Hosp. Rep.*, January, 1957.
[2] *Surgery*, December, 1956.

complications. Ochsner states that dosages required are very much in excess of those which have been used in the treatment of thymic hyperplasia; it is not beyond reason to expect that carcinogenesis will occur within a much shorter period of time, and that, instead of an average of 6·9 years as occurred in children after thymic irradiation, the period of time may be considerably shortened. It is therefore questionable whether the use of radioactive iodine is ever justified in the treatment of benign thyroid conditions, except in the patient for whom other forms of therapy are contraindicated, or in the individual whose life expectancy is so short that the possible carcinogenic effect of the radioactive material may not occur. The facts brought forward certainly add weight to the argument that radioactive substances should be kept in the hands of those who understand them and appreciate the dangers that lie in their careless or uninformed use.

A NEW JOURNAL OF MEDICAL HISTORY.

MEDICAL HISTORY is coming to be more and more accepted as a valuable part of medical knowledge. Much can be added to the understanding of disease processes, to the assessment of forms of treatment, and to a right attitude towards the patient if we know what others have done and thought in the past, whether that past is remote or recent. For this reason we welcome a new quarterly journal, *Medical History*, which is devoted to the history and bibliography of medicine and the related sciences. It is published by William Dawson and Sons Limited, 4 Duke Street, Manchester Square, London, W.1, and the annual subscription rate is £2 10s. (sterling). The first issue, that of January, 1957, contains five main articles. Under the title "Blue Books", Sir John Charles has gathered together some remarkable medical material from the vast mass of Parliamentary Papers issued in the past century and a half; Archibald L. Goodall discusses the health of James the Sixth of Scotland and First of England; S. T. Anning examines the historical aspects of venous thrombosis; Ernest A. Gray writes on John Hunter and his associations with veterinary medicine and especially with the Royal Veterinary College in its early days; Raymond Williamson describes the effect of plague on Cambridge in the fourteenth century, when it first came as the Black Death, and in the subsequent three centuries. In a section on "Texts and Documents", which is appropriately illustrated, there are descriptions of a copy of George Armstrong's printed proposals for establishing the Dispensary for Sick Children in London in 1769, by F. N. L. Poynter, and of a seventeenth-century Oxford licence to practise medicine, by K. F. Russell, of Melbourne. The journal also contains reports of societies concerned with the history of medicine, general notes and news, book reviews and a list of medical anniversaries of 1957 set out according to the appropriate month. This is an attractive journal, and we hope that it will long flourish.

KLEBSIELLA PNEUMONIÆ.

SINCE the early nineteen-thirties, for one reason or another, but partly because of antibiotics, a great deal of the sting has been removed from pneumonia. A good prognosis can usually be given. However, one of the remaining stumbling blocks is pneumonia due to Friedländer's bacillus. This cause of pneumonia is probably commoner than figures indicate, since many people probably die quickly, and no post-mortem examination is made, and quite often Klebsiella enters the stage of pneumonia, unknown, as a last act. That this is so is becoming evident from the experience of clinicians with pneumonia in older people. With them it is not uncommon to find that the chest infection has by the third week become a cavitating lung with Klebsiella, and all is accomplished silently. The mortality from this form of pneumonia continues to be severe, and even with survival the morbidity is considerable, with much residual damage. L. P. Jervey and M. Hamburger[1] have reviewed 30 cases of acute primary Friedländer's bacillus pneumonia, 15 occurring before the advent of streptomycin, and 15 occurring after the advent of streptomycin. In the cases occurring before streptomycin became available 11 of the patients died—a mortality of 73%. All the survivors received sulphonamides, but at least one was left with residual damage. In the "post-streptomycin" group of 15 cases the patients were treated with varying combinations of streptomycin, penicillin, tetracycline drugs and sulphonamides. Eight of the 15 patients died. In five of the eight death occurred within forty-eight hours. Of the survivors the "follow-up" was most unsatisfactory, but once again, as far as can be ascertained, residual lung damage is common. In-vitro sensitivity testing showed that every strain of Klebsiella isolated was resistant to penicillin and sensitive to streptomycin.

The figures in this series are too small for definite conclusions, as with most series on Klebsiella pneumonia; but it does seem that the mortality has decreased slightly. The mortality remains very high, however, and residual damage seems equally important. What is probably a most important factor is early diagnosis, as four of the patients in the post-streptomycin group died quickly before a diagnosis was made, Klebsiella being found *post mortem*. Diagnosis may need to be over-eager and made in the absence of evidence, in terms of bacteriology or physical signs, if lives are to be saved.

THE VITAMIN B_{12} CONTENT OF HUMAN LIVER.

SINCE the introduction of methods of estimating vitamin B_{12} absorption by means of radioactive cobalt, it has become known that many gastro-intestinal disturbances will reduce absorption greatly. Lack of absorption of vitamin B_{12} has been associated with pernicious anæmia, partial and total gastrectomy, steatorrhœa, old age and intestinal shunts and loops. It is noteworthy that after operative interference to the gut, as, for instance, after a total, or high partial, gastrectomy, it may be three years or more before the development of macrocytic anæmia. This would indicate that stores of vitamin B_{12} are probably large. It has been recorded, for instance, by S. O. Schwartz and H. Legere[2] that when patients with pernicious anæmia stop their therapy, it may be four years before a relapse occurs.

M. E. Swendseid *et alii*[3] analysed liver tissue obtained at autopsy in 132 males. One cause of death, cirrhosis of the liver, was considered separately. None of the subjects had taken vitamin B_{12} parenterally. They found that liver kept in an icebox for periods of up to seven days showed no deterioration of vitamin B_{12} content. By arrangement of the subjects into three age groups (twenty to forty, forty to sixty, and over sixty years of age), the liver content of vitamin B_{12} could be noted in relation to the supposition that decreased absorption of vitamin B_{12} occurs in old age. The average amounts of the vitamin in the three groups were 0·72, 0·73 and 0·71 microgramme of vitamin B_{12} per gramme of wet liver tissue. The average amount for the subjects with cirrhosis of the liver was 0·26 microgramme of vitamin B_{12} per gramme of wet liver tissue. These figures indicate that the ability of a cirrhosed liver to store vitamin B_{12} may be impaired.

If the average adequate daily requirement of a human being for vitamin B_{12} is accepted as being one microgramme, and the average total weight of the human liver as 1500 grammes, then the average value of 0·7 microgramme per gramme of wet liver tissue represents a three-year store, if all the vitamin is available to the body.

These experiments should considerably advance our understanding of the role of vitamin B_{12} in disease, particularly in diseases in which the liver is suspected of poor function.

[1] *Arch. Int. Med.*, January, 1957.
[2] *J.A.M.A.*, March 4, 1944.
[3] *Blood*, January, 1957.

Abstracts from Medical Literature.

PHYSIOLOGY.

The Pulmonary Alveolar Moisture.

C. C. Macklin (*Dis. Chest*, September, 1956) describes how the thin film of mucoid fluid of low surface tension, which moistens the walls of the pulmonary alveoli, is produced. He states that the granular pneumonocytes, which lie free on the alveolar walls, contain particles which can be seen by oil-immersion phase microscopy at the limit of visibility, looking black with the dark medium lens. These particles are hygroscopic and deliquescent and take up moisture from the alveolar air, swelling up in the process to become spheres enclosed in an elastic pellicle. The moistening of the inspired and expired air is effected not in the alveoli but in the air passages.

Experimental Motion Sickness in Dogs.

S. C. Wang and H. I. Chinn (*Am. J. Physiol.*, June, 1956) report that motion sickness was experimentally induced in dogs by means of a standardized swinging exposure. Susceptible dogs were selected for surgical extirpation of the labyrinths or various parts of the cerebellum. It was found that animals showed no vomiting responses to long exposures of swinging motion after bilateral labyrinthectomy or ablation of the nodulus and uvula. Even with incomplete extirpation of these structures, animals would become partially or totally resistant to motion sickness. In general, these animals exhibited normal responses to intravenously administered apomorphine or orally administered copper sulphate. These results indicate that motion stimulates the labyrinthine receptors, and the vestibular impulses traverse the nodulus and uvula of the cerebellum, and the chemoceptive emetic trigger zone, and finally reach the medullary vomiting centre.

Temperature Gradients in the Hypothermic Dog.

G. B. Spurr, S. M. Horvath, L. H. Hamilton and B. K. Hutt (*Am. J. Physiol.*, July, 1956) report that temperature gradients have been studied in 15 dogs subjected to hypothermia. In six of these experiments it was possible to maintain the rectal temperatures at approximately 25° C. for four and one-half to thirty-four hours. The results obtained indicate that there was little extraction of heat from the skin during progressive hypothermia. Initially the greatest amount of heat lost to the environment was from the muscular tissue. As the hypothermia progressed a greater and greater flow of heat came from the deep central regions, and less and less of the heat lost to the environment originated in the muscular tissues. Throughout the period of stable hypothermia the temperature gradient between the core (rectum) and the muscular tissue of the thigh was significantly greater than during the control period; this suggested that the flow of heat depended primarily on conduction. The values for the "thermal circulation index" of the hind footpad, thigh and foreleg were found to decrease as a result of hypothermia, while the index for the ear and chest showed no significant change. These results, together with the evidence on the temperature gradients, indicate that the induced hypothermia resulted in an increased volume of the "shell" and a consequently reduced volume of the "core". Since there was no appreciable change in the index for the chest, a relatively greater proportion of the heat lost to the environment must have occurred from the surface of the trunk.

Age Changes in Body Size, Body Composition and Basal Metabolism.

M. C. Conrad and A. T. Miller (*Am. J. Physiol.*, August, 1956) report that the interrelations of body size, body composition and basal metabolism were studied in 69 albino rats ranging in age from eighteen to one hundred and seventy-four days. The decline in metabolic rate with age was more rapid than would be predicted from the weight$^{0.57}$ rule which eliminates the influence of body size in interspecific measurements. Body composition analyses indicated that the increase with age in metabolically inert fat and bone minerals was approximately balanced by a corresponding decrease in metabolically inert extracellular fluid, so that "active tissue mass" was virtually unchanged. Calculations based on data in the literature indicate that about one-half the decline in metabolic rate with age may be due to the corresponding decrease in the relative weight of the viscera. The remainder of the decline in metabolic rate must be due to factors other than changes in the chemical or histological composition of the body.

Role of the Spleen in Acclimatization to Hypoxia.

S. F. Cook and M. H. Alafi (*Am. J. Physiol.*, August, 1956) report that to determine quantitatively the participation of the spleen and the bone marrow separately, five groups of splenectomized and non-splenectomized mice totalling 87 individuals were exposed to a simulated altitude of 15,000 feet continuously for periods of thirty to fifty-eight days. Red blood cell counts and hæmatocrit determinations were made at various intervals. It was found that about two-fifths of the increase in red cells could be referred to a tonic contraction of the spleen and the remaining three-fifths to the production of red cells by the bone marrow.

Some Effects of Ergotamine Tartrate Upon Lactation in the Rat.

C. E. Grosvenor (*Am. J. Physiol.*, August, 1956) reports that a study was made of the direct effects of ergotamine tartrate upon the lactating rat and her young, and upon milk secretion from her mammary glands. The drug was administered to lactating rats at a dosage of three milligrammes per kilogram per day on each of the ninth to twelfth post-partum days. It was found not to affect the volume of milk in the mammary gland as measured by lactose content of mammary tissue on the twelfth day, nor was it found to have altered the histological appearance of the gland. There is, however, significant loss in weight of the mothers during the period of treatment correlated with significant reduction in food consumption. Recovery in weight occurs by the end of lactation. There is also significant weight loss of litters of treated rats during the same period, although they suck more frequently during the period of treatment than do controls. The possible significance of this is discussed.

Rate of Elimination of Labelled Carbon Dioxide from the Body.

D. R. Drury, A. N. Wick and M. C. Almen (*Am. J. Physiol.*, August, 1956) report that when C^{14}-labelled bicarbonate is injected intravenously about 10% of it is exhaled within thirty seconds by the lungs before it is mixed with body carbon dioxide. Labelled carbon dioxide that has escaped elimination by the lungs is mixed with body carbon dioxide in a complex manner. The carbon dioxide in those tissues with a high blood flow equilibrates very rapidly with that of the blood. Resting muscle and skin need at least five minutes for equilibration with the carbon dioxide of the blood. Calculations of body carbon dioxide pool based on the assumption of complete instantaneous equilibrium between blood and tissues are not valid. The manner of elimination of labelled carbon dioxide produced in metabolism is complex and does not simulate that following intravenous or intraperitoneal injection of labelled bicarbonate.

The Effect of Digoxin on the Kidneys.

A. L. Hyman *et alii* (*Am. Heart J.*, October, 1956) injected digoxin into the renal artery of normal dogs and of a dog with congestive heart failure. There were appropriate controls. A direct effect was demonstrated of digoxin on the kidney, leading to the excretion of sodium and water; this resembled the effect of mercurial diuretics, except that there was no increase in potassium excretion.

Hypothermia in Hæmorrhagic Shock.

E. W. Friedman, D. Davidoff and J. Fine (*Am. J. Physiol.*, June, 1956) report that precooling to 28° C. alters the course of hæmorrhagic shock in the dog by prolonging the period of tolerance to severe hypotension, and by prolonging the survival time after transfusion from an average of several hours to an average of thirty hours. Precooling does not prevent death; but if an antibiotic is given at the time of transfusion, all precooled dogs recover, whereas normothermic dogs given an antibiotic at the time of transfusion do not recover. Precooling protects the dog in hæmorrhagic shock by sustaining the antibacterial defence mechanisms, which disintegrate rapidly in the normothermic dog. The protective effect of cooling is not secured if it is applied after the induction of hæmorrhagic shock. The effects of hypothermia upon the cardio-vascular dynamics are described, and their significance is discussed.

BIOCHEMISTRY.

Glucagon.

N. KALANT (*Arch. Biochem.*, December, 1956) has reported on experiments in which fasted intact rats were injected with glycine-1-C^{14} and glucose-1-C^{14}, and the effects of glucagon and adrenaline on the incorporation of the C^{14} into liver glycogen and expired carbon dioxide were studied. The rate of incorporation of C^{14} from glycine into liver glycogen was increased by a previous injection of glucagon and decreased by a previous injection of adrenaline. The rate of incorporation of glycine-1-C^{14} into glycogen was decreased, while the incorporation into carbon dioxide was increased by concurrent injections of glucagon. The incorporation of C^{14} from glucose into liver glycogen was increased by a previous injection of glucagon. Glucagon appears to cause a "rebound" accumulation of liver glycogen comparable to that produced by adrenaline.

Lipæmia.

H. ENGELBERG (*J. Biol. Chem.*, October, 1956) has demonstrated endogenous lipolytic activity, as determined by the release of free fatty acids or glycerol *in vitro*, in the plasma of nine normal individuals without the prior injection of heparin. Inhibitor studies indicate that human endogenous lipæmia-clearing factor (lipo-protein lipase) is similar to or identical with the post-heparin-clearing factor. Its inhibition by protamine indicates that heparin is probably a component. The results afford further evidence of the biochemical role of this factor in lipoprotein transformations in the blood-stream.

Sulphates.

J. J. SCHNEIDER AND M. L. LEWBART (*J. Biol. Chem.*, October, 1956) have incubated 32 steroids in a system consisting of microsome-free supernatant fluid of rabbit liver, adenosine triphosphate and sulphate and magnesium ions. Evidence was obtained that 14 of these steroids, including testosterone and desoxycorticosterone, were conjugated with sulphuric acid in this system.

Salicylic Acid.

R. PENNIALL *et alii* (*Arch. Biochem.*, October, 1956) have studied the compounds aspirin, salicylic acid, gentisic acid and salicyluric acid for their effects on in-vitro brain respiration. Each of the compounds tested was found to inhibit in-vitro brain glycolysis. Salicylic acid was found to uncouple oxidative phosphorylation from oxygen uptake during the oxidation of pyruvate by both homogenates and mitochondria of brain tissue. A similar effect was observed for salicylic acid on the oxidation of succinic acid by brain mitochondria. Salicylic acid was also inhibitory to the oxygen uptake of succinate oxidation, but this effect was observed only at concentrations two to three times the levels necessary for demonstration of the uncoupling action. Aspirin, gentisic acid, and salicyluric acid were found to inhibit the aerobic respiration of brain mitochondria. However, these effects were noted only at concentrations ten times that necessary for demonstration of salicylic acid. The uncoupling action has been found to be reversible.

Sterols.

W. W. WELLS (*Arch. Biochem.*, January, 1957) has studied the formation of copostanol. The addition of sodium taurocholate to fat-free diets containing 0·25% cholesterol led to significant inhibition of copostanol formation. "Tween 80" produced similar results. Complete absence of bile had no effect on the extent of copostanol formation. The addition of sodium taurocholate and "Tween 80" to cholesterol diets enhanced the cholesterol absorption as indicated by increased blood and liver cholesterol levels, and a corresponding decrease in total sterol excretion characterized by depressed copostanol and increased cholesterol excretion. The amounts of the Δ^7 sterols excreted increased with age, but were not greatly affected by the presence of cholesterol or of the emulsifiers in the diet.

D. L. COLEMAN AND C. A. BAUMANN (*Arch. Biochem.*, January, 1957) have continued an investigation of intestinal sterols. Male rats excreted more sterol than females; castrates excreted less of all sterols except copostanol. The ratio of Δ^7-cholesterol to cholesterol excreted increased greatly with age. Penicillin and succinyl sulphathiazole depressed the excretion of copostanol with corresponding increases in cholesterol. "Aureomycin", streptomycin and "Chloromycetin" were without effect. Sterol excretion was essentially the same on a fat-free diet as on a diet containing 20% of lard. It was increased when cellulose was fed. Cholesterol markedly increased the excretion of copostanol; squalene and Δ^7 cholesterol did not. Δ^4-cholesterone increased the excretion of each of the five sterols measured.

Protein Breakdown.

D. STEINBERG AND M. VAUGHAN (*Arch. Biochem.*, November, 1956) studied protein degradation in rat liver and rat kidney slices using two methods: (i) measurement of the release of radioactive amino acids from tissues previously labelled *in vivo*; (ii) measurement of net changes in the non-protein nitrogen fraction. Protein breakdown, measured by either method, was found to be strongly inhibited by anaerobiosis, by dinitrophenol and by certain amino-acid analogues. The properties of the protein breakdown observed suggest that in mammalian tissues there is a catabolic phase of protein metabolism and that there is, at the cellular level, a true dynamic state of proteins. Some of the other implications of the inhibitor effects are discussed.

Succinic Dehydrogenase.

T. P. SINGER *et alii* (*J. Biol. Chem.*, December, 1956) have isolated succinic dehydrogenase from heart mitochondria as a soluble protein in a state approaching homogeneity by physico-chemical criteria. The overall purification is about one hundredfold compared with a mitochondrial acetone powder. The enzyme is a ferroflavoprotein containing four atoms of ferrous (non-hæmin) iron and a mole of flavin per mole of protein (200,000 grammes). At 38° C. the optimum pH is 7·7. Oxalocetate, malonate and fumarate are competitive inhibitors. Antimycin A and BAL do not inhibit the enzyme. It is highly sensitive to sulphhydryl reagents, p-chloromercuribenzoate inhibiting it in a reversible manner and the substrate protecting the enzyme from this type of inhibition.

Body Composition.

R. M. FORBES *et alii* (*J. Biol. Chem.*, December, 1956) have reported the chemical composition of two adult human bodies in terms of 14 separate tissues and organs. Data are given for water, ether extract, protein, ash, calcium, phosphorus, magnesium, boron, cobalt, beryllium and strontium. The whole bodies contained 27·93% and 4·32% of ether extract. The following major constituents of the fat-free bodies were, respectively, water 70·11% and 72·9%, crude protein 23·81% and 20·62%, ash 6·81% and 6·01%, calcium 2·15% and 2·07%, phosphorus 1·13% and 1·11% and magnesium 0·049% and 0·047%. Boron and cobalt were widely distributed, and occurred in all tissues, with highest concentrations of boron in the skeleton (0·6 to 0·9 part per million) and cobalt in the liver (0·126 and 0·064 part per million). Beryllium was found in only two tissues of one specimen (liver 0·012 and lungs 0·023 microgramme per 100 grammes). Measurable amounts of strontium were found only in the skeleton (80 and 30 parts per million).

Experimental Shock.

J. E. GUTHRIE AND J. H. QUASTEL (*Arch. Biochem. & Biophys.*, June, 1956) have shown that the rates of absorption of glucose and DL-alanine, D-leucine and L-leucine, and L-glutamine by the isolated, surviving, guinea-pig small intestine, obtained from animals that have succumbed to experimental (tourniquet) shock, are greatly reduced from the normal. The conversion of fructose into glucose is almost completely abolished in intestine after experimental shock; so that although fructose diffusion *per se* seems to be unaffected, the total amount of sugar (glucose and fructose) absorbed by the intestine in the presence of fructose is diminished by shock. The rate of absorption of sorbose is unaffected by experimental shock. The metabolic conditions set up in the isolated intestine after experimental shock resemble those obtained by normal intestine under anaerobiosis. Anoxia induced by asphyxia does not affect the absorption of glucose by the isolated intestine. Tying off the blood vessels supplying a segment of the intestine causes such metabolic changes that a cessation of active absorption of glucose by that segment of the intestine is produced. The authors conclude that during experimental shock a condition resembling that of anoxia is set up in the intestine, whereby metabolic conditions are affected. This results in a cessation of "active" absorption of glucose, fructose and amino acids.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

INTERNAL DERANGEMENT OF THE KNEE JOINT.

INTERNAL DERANGEMENT of the knee joint is a traditional rather than a generic term used to describe a miscellaneous collection of non-infective, non-arthritic conditions mainly of traumatic origin, in which the disability is produced largely by mechanical means. These conditions as a rule do not present any particular diagnostic difficulties, and it is to be hoped that the abbreviation "I.D.K.", so well established by custom, will eventually be abandoned. What should be included by the term and what should not is open to controversy, but the following will be considered: (i) acute hydrarthrosis; (ii) acute hæmarthrosis; (iii) strains and ruptures of ligaments; (iv) injuries of the menisci; (v) loose bodies in the joint; (vi) recurrent dislocation of the patella.

Examination of the Patient.

Since many of these conditions can be diagnosed with a fair degree of confidence on the symptoms alone, a carefully taken history is of prime importance. If the patient is one who is inclined to conceal essential features under a mass of gratuitous verbal superfluities, some patience in eliciting facts may be amply rewarded.

Since most of these conditions are associated with trauma, it is important to ascertain as far as possible precisely what happened at the original injury, how it was produced, what was done to relieve it at the time, and the details of any subsequent injury or painful incident. Common symptoms are pain, swelling, locking, instability, sensation of something loose in the joint, clicking and grating, and these should be inquired into in some detail. It is also necessary to ascertain details of any previous treatment and its effect, and whether anything has previously been disclosed by X-ray examination. Age, sex, occupation and the patient's sporting and recreational activities need to be considered.

The clinical examination may then be proceeded with. It is convenient to carry out most of the examination with the patient on the couch, his shoes, socks and trousers having been removed. Inspection and comparison with the opposite limb will reveal any wasting of the quadriceps or other muscles and any localized or general swelling. Any scars around the joint are noted. Active and passive movements are then tested, and the joint is examined to determine whether any abnormal movements such as abduction or adduction are present, whether it is possible to rock the tibia backwards or forwards on the femoral condyles, and whether the patella shows any abnormal mobility.

Palpation will disclose any heat and any local or general swellings, and these should be tested for fluctuation and tenderness. The joint may be tested to ascertain whether patellar tap can be elicited. The value of patellar tap as a sign is sometimes scorned, on the grounds that a fair amount of fluid must be free in the joint before the patella can be made to tap, and this is obvious enough in any case; but the test does serve to identify the presence of fluid as opposed to œdematous gelatinous swollen synovia, which often gives a false feeling of fluctuation. The knee is palpated for points of tenderness, especially over the joint line, and the joint is then palpated during active and passive movement to test for clicks and other adventitious sounds, their site and nature and how they are produced. The patient is then turned over into the prone position and the back of the knee is examined. Finally the joint is examined with the patient standing, since clicks and localized swellings not obvious in recumbency may appear only when weight is borne.

X-ray examination will reveal most chronic inflammatory and degenerative arthritic conditions which need to be considered in differential diagnosis, and it will also reveal opaque loose bodies, osteophytes, affections of the tibial tubercle and spine, and any calcification or ossification of the ligaments or menisci. Arthrography with air or contrast solutions such as "Diodone" may give additional information. For instance, by this means injuries of the menisci can sometimes be demonstrated; but interpretation requires some experience. Arthrography frequently fails to demonstrate the lesion, while on the other hand pseudo-positive findings are not uncommon, and it is therefore not recommended for routine use. However, in some cases—for instance, when the patient has had a previous operation—it may reveal whether the meniscectomy has been complete or whether the cartilage has regenerated.

In doubtful cases examinations of the blood, including estimation of the erythrocyte sedimentation rate and the Wassermann test, examination of the urine for albumin, and joint aspiration and examination of the fluid, complete the investigation.

Acute Hydrarthrosis.

Acute hydrarthrosis may occur in simple form from blows and strains. Swelling develops slowly and is at its maximum in about twenty-four hours or more. The knee may feel warm. If the joint becomes very tense it should be aspirated. It is treated by bandaging and a few days' rest followed by active exercises. Chronicity is favoured by too prolonged rest and by concomitant additional injuries, for which examination should be made. Occasionally simple synovitis may become chronic or recurrent to such an extent that synovectomy is warranted. In many cases labelled "internal derangement of the knee joint" in which symptoms of instability are present and effusions recur, the only lesion is a weak quadriceps from excessive rest and inactivity.

Acute Hæmarthrosis.

Acute hæmarthrosis may be produced in a similar manner to acute hydrarthrosis, already described, but it is more likely to be associated with other injuries to the joint. The joint swells much more rapidly and may become tensely distended in a few hours. There may be considerable pain, swelling, heat, leucocytosis and even malaise. At first the blood remains fluid and mixed with synovial fluid, the secretion of which is stimulated at the same time; but it may clot, and this should be anticipated and the joint aspirated. Otherwise the treatment consists of rest and bandaging. A splint may add to the patient's comfort. Quadriceps exercises should start in a few days, and movements and weight-bearing when the acute phase has passed and other injuries have been excluded. Clotting may produce long-continued or permanent disability.

Ruptures of Ligaments.

Rupture of the collateral ligaments may be partial (sprain) or complete. After an injury the joint may be so painful that an anæsthetic may be necessary to determine the degree of damage. The clinical features are pain, effusion and instability; pain is increased by putting the affected ligament on the stretch, and abduction or adduction is possible if the rupture is complete. Two or three weeks' fixation in plaster of Paris is by far the most efficient form of treatment for the more minor injuries, but many complete tears will heal by this means. However, if the tear is complete, exposure with suture is a more certain means of preventing chronicity. Post-traumatic calcification in the medial collateral ligament has been dignified with the name "Pellegrini Stieda's disease".

Rupture of the anterior cruciate ligament allows the tibia to be pulled forwards on the femoral condyles with the knee flexed, and rupture of the posterior cruciate ligament, which is rare as an isolated injury, allows the opposite abnormal movement. Three weeks' immobilization in plaster of Paris are required. If X-ray examination reveals avulsion of the tibial spine, it is feasible to open the joint and replace it. Chronic slackness of ligaments is common and may be associated with or confused with other injuries, such as ruptures of the menisci. Many of these could probably have been prevented by recognition and appropriate treatment at the time when the injury occurred. Slack collateral ligaments can readily be tightened by operative measures; but operative reconstruction of cruciate ligaments has not had much popularity. In some cases of chronic lax knee joints the Marsh knee cage may be of value.

Injuries of the Menisci.

Injuries of the menisci are common, and the medial meniscus is more commonly injured than the lateral. Characteristically these injuries occur in soldiers, miners and athletes, especially footballers. Whilst the medial meniscus may be injured by a twisting movement while weight is bore on the semi-flexed knee, this is not the only mechanism.

Menisci can be injured in a variety of ways, including direct trauma to the joint, traffic accidents, jumping onto the flexed knees from a height *et cetera*. Locking is a dramatic and characteristic symptom of a partial tear of

the medial meniscus, and this is accompanied by pain and followed by swelling. In the absence of a history of locking many meniscus injuries are frequently overlooked. Only a partial bucket-handle tear and the partly detached tag of meniscus are capable of causing a knee to lock. In dealing with the acutely locked knee only the gentlest of manipulation should be attempted, and if this fails, flexion, abduction and rotation should be repeated under anæsthesia with relaxants. If the joint is unlocked with the minimum of additional trauma and treated by joint immobilization, some acute injuries will heal completely. Forcible reduction, on the other hand, may unlock the knee by converting a partial bucket-handle tear into a complete one, and failure to heal is certain. The torn portion is displaced to the centre of the joint, where it will prevent full extension, but otherwise the knee may appear for a time to be quiescent. When the condition becomes chronic the symptoms include repeated incidents of painful locking followed by effusion, repeated attacks of pain and effusion without locking, intermittent or continuous local pain and tenderness, instability and clicking. Important clinical signs are quadriceps wasting, effusion, tenderness localized to the meniscus, fullness around the patellar tendon, and the elicitation of clicks by flexing and rotating the joint.

McMurray has described the following sign:

> The patient must be recumbent and relaxed, the surgeon standing at the side of the injured limb; he grasps the foot firmly and the knee is bent so that the foot approaches or touches the buttock. The foot is now rotated externally and the leg abducted at the knee whilst the joint is slowly extended. With the alteration of the angle of the joint any loose portion of the internal similunar cartilage is accompanied by a definite click and pain which the patient states is similar to the feeling experienced each time the knee gives way. The angle at which this occurs gives the position of the cartilanginous lesion, and if the manœuvre is correctly carried out the absence of such a click is a definite indication of the absence of any lesion in the posterior or middle positions of the cartilage.

The statement contained in the final sentence of the quotation is open to question. I have removed many menisci showing definite lesions from knees from which no click could be elicited by any method. On the other hand, clicks can sometimes be elicited from apparently healthy joints. The diagnosis of many injured menisci has been missed because the knee neither clicks nor has locked. The clinical evidence of injuries to the menisci depends on three features. The tear itself may produce locking, instability, clicking and limitation of extension. The chronic traumatic chondritis produced by the injury at the same time is responsible for much of the pain, the local tenderness and the swelling of the meniscus which make it abnormally palpable, and in late cases calcification may be apparent radiologically. Secondary arthritis produces intermittent or chronic synovitis with effusion, synovial hypertrophy with œdema and increased subsynovial deposition of fat, which is evident from fullness around and below the patella, and in the untreated patient the signs of osteoarthritis eventually appear. The two last-mentioned conditions may occur in the absence of any definite macroscopically evident rupture of the meniscus. There need be no misgivings on removing an unruptured meniscus which has definitely been causing symptoms, because histological examination will show that it is not normal fibrocartilage, and the patient is cured.

To recapitulate, recent injuries of the menisci in which the joint can be unlocked and fully extended can be given a trial of non-operative treatment. In acute cases in which this cannot be done, in those in which recovery is not rapid, and in all chronic cases operation is required—the sooner the better. If a patient has had an injury to the knee and has recurrent attacks of pain and swelling, if there is persistent tenderness localized to one or other meniscus, if the ligaments show no slackness and if the X-ray findings are negative, operation should be advised whether locking and clicking have occurred or not.

Some injuries of the menisci are followed by cystic degenerative changes. The cysts are multilocular and contain gelatinous material, and they may vary in size from time to time. Calcification is sometimes seen in an X-ray film. These injuries are more common in the lateral meniscus and produce a hard swelling in the joint line. Sometimes they are apparent only when the patient protrudes the swelling by bearing weight on the flexed knee. The symptoms are pain and swelling, and the treatment is meniscectomy.

"Nipping of synovial fringes" has been regarded as a clinical entity, but synovial hypertrophy is almost always a secondary condition, and it is doubtful whether these synovial villi can in fact be "nipped" by the smooth surfaces in the interior of the knee joint, though this may possibly be one factor in producing symptoms in arthritic joints.

Discoid lateral meniscus is an atavistic developmental anomaly. The meniscus may be completely circular. Characteristically it produces a loud and often easily audible "clunk"; but otherwise the condition is benign, and unless it is producing pain no treatment is necessary. Snapping tendons around the knee are sometimes due to impingement of irregular bony surfaces or to osteomata, and they sometimes suggest defects of the menisci.

Loose Bodies in the Knee Joint.

Loose bodies occur in tuberculous, arthritic and neuropathic joints; these are conditions readily recognized radiologically, and scarcely come within the scope of "internal derangement".

Fibrinous and cartilaginous loose bodies occasionally follow injury, and sometimes pieces of bone may be broken off and lie free within the joint.

The commonest cause of loose bodies within the joint between the ages of ten and forty years is *osteochondritis dissecans*. The condition is common in the knee joint and less common in the elbow, and occurs rarely in the hip, ankle and other joints. The ætiology is uncertain. It is frequently familial, and sometimes more than one joint is affected in the same patient. In some cases both knees and both elbows are involved. A piece of bone on the articular surface appears to lose its blood supply, possibly from trauma, and a sequestrum forms; this together with an area of overlying articular cartilage eventually becomes detached and shed into the joint. The area of detached cartilage is usually much larger than the piece of bone. The inner aspect of the weight-bearing area of the medial femoral condyle is the common site. It produces attacks of painful locking followed by swelling. The patient may feel something loose in the joint moving about. The condition is readily recognized radiologically. If it is recognized early before the sequestrum has separated, prolonged immobilization may result in revascularization and reattachment, but it is probably better to operate in all cases and remove the fragment. The symptoms may very closely imitate those of a ruptured meniscus, and it is therefore necessary to examine all knees radiologically before attempting to remove a meniscus, however certain the diagnosis seems. The prognosis is not particularly favourable, since recurrences are common, especially in the older age groups, and loss of substantial areas of the weight-bearing surface of the femoral condyles may eventually result in serious disability.

Recurrent Dislocation of the Patella.

If the patella becomes displaced over the lateral femoral condyle, the quadriceps is mechanically unable to extend the knee, and pseudo-locking occurs which may be painful. Many patients with this condition are aware of what happens, but some are not, and the symptoms may resemble those of an injury to a meniscus. In the examination of any knee joint, abnormal lateral mobility of the patella should be tested. In children this condition may be associated with knock knees and muscular insufficiency, and in most cases it will correct itself when the underlying conditions have been remedied. In rare cases there is a deficiency of the lateral femoral condyle. In adults it is more commonly due to trauma; cicatricial tissue pulls the patella laterally on flexion, and the medial part of the capsule of the knee is stretched or deficient. Conservative management by physiotherapy and exercises often fails, and in such cases operation is required. The capsuloplasty which I have described (Stonham, 1954) is simple and efficient, and has not the disadvantage of transferring the patellar tendon from the epiphysis to the tibial diaphysis; it is therefore applicable in all cases, irrespective of the patient's age.

F. V. STONHAM, Melbourne.

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British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Wangaratta Base Hospital on June 23, 1956.

The Early Treatment of Squint.

DR. B. A. E. HARLEY (Wangaratta) read a paper on the early treatment of squint. He said that there was still a considerable number, not only of laymen, but also of doctors, who thought that the correction of squint could be well left until the age of nine or ten years, or even later. Delayed treatment was reasonable if the criterion of cure was two straight eyes; if the criterion of cure was two straight eyes with full binocular function, then delay was likely to be disastrous. The first would produce a good cosmetic result, if it was borne in mind that the amblyopic eye tended to diverge or converge, even after it had been put straight. He hoped that he did not need to say that his criterion of cure was full binocular vision.

Dr. Harley went on to say that there were many causes of squint in children, but he intended to confine himself to the two commonest types—paralytic and accommodative. The paralytic squints were usually present at birth, and the external rectus was the muscle most commonly affected. Some restoration of function, occasionally complete, might well occur; but a more or less pronounced convergent strabismus was most frequently the result. Spontaneous cure was so rare that it was not worth waiting for. He preferred to operate on such children at the age of about fifteen to eighteen months; most affected eyes could be put straight, although some required more than one operation. With their eyes straight, the children had the incentive and the means to establish binocular function for themselves at a time when their brains were highly plastic and adaptable. Orthoptic examination as soon as they were old enough to cooperate often revealed good binocular function, and no orthoptic treatment, or only a little, was necessary. It was, of course, imperative to have anæsthesia of a high order. The operation consisted of recession, posterior to its insertion, of the internal rectus, and resection of the external rectus. One was limited in the recession of the internal rectus to a maximum of five millimetres, because more than this placed the new insertion of the muscle posterior to the equator of the globe, and gravely reduced the power of convergence, which had to be left intact. The stay in hospital was about a week, and the first day after operation found the children in apparent comfort. While a few of the children, for reasons not entirely understood, never achieved binocular vision, no harm had been done by early operation, and it was important to bear in mind the relief of parental distress, and freedom from unkind remarks of friends and relations.

Dr. Harley then said that the accommodative squint commonly started at the age of three years, and was convergent and concomitant; that meant that the angle of squint was equal, whichever eye was fixing. That was not the case with the paralytic squint. Children with accommodative squints were moderately hypermetropic, and that was one factor, and an important one, in the causation of the squint. As accommodation and convergence, though separate functions, marched together, as did swinging the arms and walking, the effort at accommodation was thought to produce the convergence. Other factors, of course, must be involved, as many non-squinters were hypermetropic. There was often a strong family history. Treatment consisted of the prescription of spectacles to reduce the angle of squint by 10° or 15°, occlusion of the good eye if amblyopia was present, orthoptics and operation. If the angle of the squint was more than 15°, operation was indicated. Dr. Harley said that in that case he liked to operate early, as soon as equal vision in both eyes had been achieved by the use of occlusion. If such patients were left untreated, amblyopia in the squinting eye would most surely develop, and restoration of vision became more and more difficult and unlikely after the age of eight years. Orthoptic treatment became increasingly unrewarding as the habit of monocular vision was year by year more firmly established. Again, one had to bear in mind the social effects—the cruelty of the child's own little friends, few of whom would fail to make capital out of his deformity, and also the undoubted fact that affected children were almost always strikingly more manageable after successful treatment.

Dr. Harley finally said that, in common with many ophthalmologists, but not with all, he had come to the firm conclusion that there was everything to be gained by the earliest possible treatment of squint, and he had yet to discover that anything was to be lost.

A Case for Diagnosis.

DR. D. D. BROWNE (Wangaratta) showed a girl, aged ten years, who had been first examined on March 20, 1956, with a history of four weeks' lassitude and variable pains in the neck, wrists and ankles, which often awoke her at night. The ankles swelled occasionally. The onset was acute, and followed hay fever. On examination, she was seen to be a slight, pale, black-haired girl; her temperature was 97·8° F., and her pulse rate was regular at 96 per minute. Ear, nose and throat examination revealed no abnormality. The apex beat was in the fifth intercostal space in the nipple line, and there was a short pre-systolic and systolic bruit at the apex; the lungs and abdomen were normal; the reflexes were equal and active; there was slight swelling of both ankle joints, with no pain on movement. The hæmoglobin value was 12·8 milligrammes per 100 millilitres; the erythrocytes numbered 4,470,000 per cubic millimetre and the leucocytes 12,000 per cubic millimetre (76% were neutrophile cells and 12% metamyelocytes); the erythrocyte sedimentation rate (Westergren) was 31 millimetres in one hour.

A diagnosis of acute rheumatic fever was made and "Regaspirin" (10 grains) was administered every four hours, with the usual nursing.

On March 28 the heart had a slight systolic apical bruit, aching and swelling had ceased, and the sedimentation rate was 21 millimetres in one hour. On April 3 the heart sounds were normal, and the sedimentation rate was 10 millimetres in one hour. She was transferred home to continue rest in bed and treatment.

On April 18, when she was due for further examination, she developed chickenpox, but was otherwise well. Continued rest was ordered until the attack had passed, and the reporting of any fever, sore throat or other disorder was requested.

On April 25 she was sitting in bed drinking a glass of water when she became unconscious, twitching commenced in the left arm, and she could not speak. Her breathing was not affected. A colleague was staying near by on holiday, and fortunately soon attended her. An ambulance was sent equipped with oxygen and a sucker, and brought her to hospital about two hours later, with a most comprehensive and helpful letter from the doctor. He reported that fifteen minutes after losing consciousness, she was sitting in bed with normal muscular tone, and conjugate deviation of the head and eyes to the left. There was no other localizing sign. She was unconscious and vomiting. All tendon reflexes were normal and equal, and both plantar reflexes were of the Babinski type. Ten minutes later she had a left Jacksonian seizure involving the face alone, then four or five seizures, left and right. She then became opisthotonic, and had a generalized fit. The coma lightened, and she coughed and responded to painful stimulus. Five minutes later she returned to coma. The pupils were widely dilated and equal and reacted to light, contracting when the coma lightened. The pulse rate was about 145 per minute; the blood pressure was 140 to 150 millimetres of mercury, systolic. The coma was flaccid, there were no localizing signs, and the right ear drum was reddened. Subarachnoid hæmorrhage seemed likely. There was a family history of epilepsy.

During the journey, and on her admission to hospital, she was in flaccid coma, punctuated by left-sided fits every five to ten minutes. Her pulse rate was 180 per minute, her temperature was about 101° F., and the pupils were widely dilated and reacted slightly to light. The heart was not enlarged, its rate was 180 per minute and regular, and no murmurs were detected. A few purulent chickenpox blebs were noted on the abdomen. Five convulsive seizures then occurred in one and a half minutes, and the breathing became stertorous. After these, all tendon reflexes were found to be present; the left plantar reflex was of the Babinski type, and the right was normal. Urine was passed, fortunately into a clean dish, and blood was with difficulty withdrawn from a median cubital vein. It was decided to attempt lumbar puncture. This was done, with the results recorded later.

The left-sided seizures recurred every ten minutes for two hours, slowly becoming mixed, then right-sided. The tendon reflexes became weaker and disappeared, and grinding of the teeth and turning from the light were noted. There was no neck retraction. At three hours, there were right-

sided twitchings but no more seizures. By this stage two grains of phenobarbital had been given by intramuscular injection, and crystalline penicillin (1,000,000 units) was given, the dose being repeated every four hours. At three and a half hours the patient was sleeping peacefully, with occasional right-sided twitchings. The left pupil was dilated and reacted slightly to light; the right was normal. There was no deviation of the eyes. The fundi were examined and appeared normal; no hæmorrhages were seen. The pulse rate was 106 to 120 per minute, the blood pressure was 120 millimetres of mercury, systolic, and 70, diastolic, the temperature was 99·2° F., and the respirations numbered 26 per minute. The lungs were clinically clear, the heart sounds were regular with no bruits, the abdomen had no apparent tenderness or mass, and the spleen was not palpable. In the limbs all the reflexes were present though sluggish, and the plantar responses were equivocal.

At ten hours, noise and light caused thrashing about to both sides, and the patient uttered occasional hoarse cries. Movements of the right arm were becoming more pronounced. The intramuscular injection of two grains of phenobarbital was repeated.

At fourteen hours, the patient was in a general attitude of flexion with some thrashing. Irritability became extreme. The pupils were equal and reacted normally. Sixteen ounces of urine was passed, the first for eleven hours. Three cubic centimetres of paraldehyde were given intramuscularly, and soon quiet sleep followed.

At twenty-two hours the patient recognized her mother, took a sweet drink, and then slept.

At twenty-five hours she awoke with no headache and spoke rationally, but had no recollection of the last forty-eight hours. Her pulse rate was 86 per minute and regular. Her temperature (taken hourly) was 99·2° F., having reached a peak of 100·8° F. Her progress was steady and uneventful. The spleen did not become enlarged, and no petechiæ were found.

Dr. Browne then discussed the clinical pathology. He said that examination of the first blood sample revealed a leucocyte count of 37,000 per cubic millimetre, 80% being polymorphonuclear cells and 15% metamyelocytes. The erythrocyte sedimentation rate (Westergren) was 38 millimetres in one hour, and the blood urea content was 172 milligrammes per 100 millilitres. Culture eventually produced a weak growth of *Streptococcus viridans*. The urine specimen passed early contained one-quarter albumin, no acetone or diacetic acid, and numerous erythrocytes and granular casts; attempted culture of this and a later specimen produced no growth of organisms.

At lumbar puncture the cerebro-spinal fluid was surprisingly not under pressure, not obviously blood-stained, and not purulent. Microscopic examination of the fluid revealed 300 erythrocytes per high-power field, thought to be of traumatic origin, and three lymphocytes. The protein level was 25 milligrammes per 100 millilitres, and the chloride content 750 milligrammes per 100 millilitres. No organisms were seen on Gram staining, and attempted culture produced no growth of organisms.

Further tests revealed rapid disappearance of albumin from the urine, and on May 1 (after six days) it contained epithelial cells and a few pus cells. On that date, the erythrocyte sedimentation rate was down to 10 millimetres in one hour, and the leucocyte count was 11,000 per cubic millimetre.

On May 7 the urine contained a few granular casts, but there was no other abnormality. The erythrocyte sedimentation rate was 12 millimetres in one hour, the leucocyte count was 9000 per cubic millimetre, and the blood urea content was 50 milligrammes per 100 millilitres. On May 14 the urine was normal, the blood cells were normal, and the erythrocyte sedimentation rate was seven millimetres in one hour, and on June 18 full blood and urine examination gave normal results.

Penicillin treatment ceased on May 14, and treatment with sulphadimidine had been continued, the dosage at the time of the meeting having been reduced to 0·5 gramme twice a day.

Dr. Browne said that he had presented the case as a problem in diagnosis, considering particularly abnormal presentation of bacterial endocarditis and acute pyæmia with pyelonephritis.

Congenital Absence of One Lung.

DR. M. ROHAN (Wangaratta) first showed a boy, aged seven years, with congenital absence of one lung. Dr. Rohan said that he was shown because of the rarity of the condition, and because the case illustrated how well adjusted the body became to such an abnormality. The boy appeared healthy. The diagnosis had been first made when he was aged five months; he had on that occasion been admitted to the Royal Children's Hospital because of pneumonia which would not respond to penicillin. X-ray examination at that time revealed dextrocardia, the apex beat being palpable in the right axilla. At the age of eighteen months, a bronchographic examination revealed complete absence of the right bronchus. On examination of the child, it was obvious that the cardiac impulse was in the right axilla, and on deep breathing it was evident on inspection that the left side of the chest moved more than the right side. Auscultation revealed the presence of breath sounds in all areas. The child was well and healthy.

Congenital Heart Disease.

Dr. Rohan then showed two patients with congenital heart disease; he said that the two cases occurred within three months of each other.

The first patient was a female, born on December 11, 1955; her abnormality was first diagnosed at the age of seven weeks, when she was having feeding difficulties. She was being artificially fed, but was not gaining weight satisfactorily and was not taking sufficient food. When she was examined, a definite cyanotic tint was present in nose and lips and the child was very dehydrated and ill looking. The only cardiac abnormality was a systolic bruit. No cyanosis had been detected at birth. Angiocardiographic investigation at the Royal Children's Hospital revealed a condition of transposition of the great vessels; it was apparent that the dye entered the right auricle from the superior *vena cava*, then traversed the right ventricle and was extruded immediately into the aorta. The child was still progressing, though recently she had begun to be exhausted more easily. Her weight at birth was six pounds fourteen ounces, and her present weight was eleven pounds five ounces. She was being fed on lactone syrup. Cyanosis and pulmonary vascular congestion were characteristic of that malformation. The only manner in which oxygenated blood might reach the tissues was by way of shunts allowing cross-mixing between the two circulations prior to injection through the transposed aorta. The ideal correction of that condition would lie in the retransplantation of the great vessels to their normal anatomical locations; but as the coronary vessels arose from the systemic trunk, that would entail their partial removal along with the proximal part of the aorta. When a large ventricular septal defect accompanied the transposition, the patient tended to survive longer. When no such defect existed, life was sustained for a time by admixture of blood through the *foramen ovale*, auricular septal defects if present and a patent *ductus arteriosus*. If there was associated pulmonic stenosis or hypoplasia, the main life-line was via enlarged bronchial arteries from the aortic trunk, the anastomosis being through the *vasa vasorum* supplied to the pulmonary arteries by the bronchial arteries. As the technique of retransplantation was difficult, attempts to improve the condition had been made in two ways: (i) by creation of a communication between the aorta and the pulmonary artery close to the coronary ostia; (ii) by the production of an artificial communication between the right atrium and the pulmonary veins (Blalock), where an anastomosis had been formed between the right pulmonary veins and the posterior surface of the right atrium. That was augmented by an anastomosis between the right subclavian artery and the right pulmonary artery.

In the second case of congenital heart disease the condition had been diagnosed at birth, because cyanosis was present. The patient was a male child born in March, 1956, his weight at birth being eight pounds four ounces; his present weight was eleven pounds ten ounces. Electrocardiographic investigation at the Royal Children's Hospital revealed left ventricular preponderance. No bruits were audible in the heart. The diagnosis made was that of tricuspid atresia. The cyanosis was variable even when the child was at rest. There had been three or four episodes of what appeared to be an attack of pulmonary œdema with frothy discharge welling up into the mouth and nose. Dr. Rohan said that the child's life depended upon an auricular septal defect and a patent *ductus arteriosus* or an anomalous vessel supplying blood to the lung. If the ductus closed, the severity of symptoms and distress increased. The surgical procedure required was the shunt operation; in the very young the pulmonary artery-aorta anastomosis (Potts Smith) was most beneficial. It might be established as early as the eighth day of life, provided that the size of the baby was adequate (seven or eight

pounds). The pulmonary artery might be non-functioning in association with a non-functioning right ventricle, or if there was an interventricular septal defect as well, then a partially functioning pulmonary artery might be present.

The Treatment of Preeclampsia.

DR. A. R. LONG (Melbourne) read a paper on the treatment of preeclampsia, which he said was a complication of pregnancy characterized by hypertension, œdema and albuminuria. While the ætiology remained unknown, knowledge of the predisposing factors and the natural course of the disease was steadily accumulating. With that increasing knowledge, more rational measures of prevention and treatment were possible. Nevertheless, preeclampsia and its sequel eclampsia remained important causes of maternal and fœtal mortality. Nowadays, with the maternal mortality rate very low, the emphasis in obstetrics was passing to fœtal salvage. The milder forms of preeclampsia, while not seriously endangering the mother's life, could cause fœtal death. Thus the modern emphasis in the treatment of preeclampsia was prevention.

Dr. Long went on to say that the prevention of preeclampsia was one of the main objects of ante-natal care. Special watch had to be kept on patients with predisposing conditions, such as essential hypertension, obesity and multiple pregnancy. The first warning of impending preeclampsia might be abnormal fluid retention without frank œdema. That was due to pathological retention of sodium in the body and showed as excessive weight gain. The normal weight gain in pregnancy was about 24 pounds. The accepted maximum total weight gain in any one month was three pounds. However, excess weight gain was not necessarily due to retained water. Deposition of fat or increasing obesity was the alternative cause. Treatment should be aimed at both those causes. The patient was given a diet of high protein content, and low carbohydrate and fat contents. The daily caloric intake should be restricted to 1700. Most important was the reduction of sodium intake by prohibiting the addition of salt to food either in cooking or at the table. Digestive powders containing sodium salts were likewise forbidden. The use of diuretics such as ammonium chloride was perhaps not necessary in the absence of œdema. The evaluation of weight control on its own was difficult, because in all published series it had been only one of the factors rigorously pursued in the prophylaxis of preeclampsia. Also failure to gain weight and actual weight loss in themselves were now considered to be early signs of placental insufficiency. Thus in practice weight gain was not always easy to interpret. More important was the recording of the blood pressure. In pregnancy the upper limit of normal was frequently stated to be 140 millimetres of mercury, systolic, and 90 millimetres, diastolic. Those figures were probably too high. From a prophylactic point of view a relative rise was more important than the actual level of the blood pressure. If a rising blood pressure was treated early by strict rest, mild sedation, salt restriction and diet as outlined earlier, then there was an excellent chance that the condition would develop no further. Perhaps the best sedative in those early cases was phenobarbitone, given in doses of half to one grain three times a day. Rest at home was not always easy to enforce, and unless improvement was rapid, early admission to hospital was desirable for either excessive weight gain or rising blood pressure. That created economic difficulties in the home and a strain on hospital beds. It was better to use beds for prophylactic treatment early than to use the same beds at a later time to treat severe preeclampsia. In the early cases, in which albuminuria had not developed, the patient might be allowed to go home when her condition had improved, and the pregnancy might be carried on under strict supervision (weekly visits). While induction of labour had little place in the management of those patients, it might be unwise to allow the pregnancy to continue beyond term, particularly in a *primigravida* aged over thirty years.

Dr. Long then discussed the severe case. He said that, with the emphasis now on prevention, it was reasonable to consider as severe any case of preeclampsia in which albuminuria developed. If further classification was desired, then the term "imminent eclampsia" might be applied to all patients with preeclampsia with symptoms who had a blood pressure over 160 millimetres of mercury, systolic, and 100 millimetres, diastolic, and whose urine contained more than "one-half" albumin. Once albumin had appeared in the urine, admission to hospital was imperative, and the patient should not be allowed home again before delivery. Strict rest in bed, preferably in a quiet single room, was essential. Sedation might be effected by the oral administration of phenobarbitone, or in the more severe cases three grains of sodium phenobarbitone might be given by intramuscular injection three or four times a day. In cases of moderate severity a protein-rich diet as outlined earlier was satisfactory; but the gravely ill patient was unlikely to take more than a light fluid diet. Salt restriction was again most important. The keeping of strict fluid-balance charts was a great help in ordering fluid intake. Little harm could be done by giving an amount equal to the urinary output *plus* about two pints a day to cover insensible loss. In the presence of a diminished urinary output it was wrong to force fluids. The use of hypotensive drugs in preeclampsia had proved rather disappointing. It was not easy to assess the fall in blood pressure due to any drug when, with rest and sedation alone, there was usually a considerable improvement over the first forty-eight hours. It now appeared that, even if those drugs did lower the blood pressure, they achieved little as far as urinary findings were concerned. Hypertension in preeclampsia was a result rather than a cause. It might be argued that merely reducing the blood pressure without relieving renal arteriolar spasm could be dangerous. The use of hypotensive drugs would appear to be reasonable if the blood pressure was alarmingly high and serious accidents, such as cerebral hæmorrhage, were feared. Various drugs might be used. Of the ganglion-blocking agents, hexamethonium bromide given by injection had had extensive trials. A test dose of 20 milligrammes was given, and the administration was repeated two hourly, the dosage varying according to response. "Serpasil" given by injection was probably safer, though it acted more slowly. Two to three milligrammes were given initially, and further doses were given every six to eight hours as necessary. "Veriloid", an alkaloid of the "Veratrone" group, had received favourable comment in the overseas literature. In the treatment of "imminent eclampsia", additional measures for the control of convulsive seizures might be employed. Morphine in a dose of one-quarter of a grain was given once and not repeated. Magnesium sulphate in a 50% solution was given by intramuscular injection. The dose was 10 millilitres given initially and then two millilitres every four hours.

Dr. Long then discussed the management of the pregnancy. He said that after the patient's admission to hospital and the commencement of treatment, preeclampsia usually underwent a pronounced remission. That was not invariable, as some patients, particularly those previously treated to some extent at home, became steadily worse. If albuminuria had developed, improvement under treatment tended to be temporary, and then, despite all therapy, the preeclampsia returned and progressed. Before termination of pregnancy was discussed, the risks of carrying it on should be considered. Fulminating preeclampsia or preeclampsia not controlled by treatment was likely to progress to eclampsia with its attendant dangers to both mother and fœtus. Even without the occurrence of convulsions there was a definite maternal mortality in such cases. Although the question was not finally settled, there was probably little risk of permanent renal damage or residual hypertension due to preeclampsia, even if albuminuria had been allowed to continue for some time. It was argued that preeclampsia was more common, not only in patients with essential hypertension, but also in those who were likely to become hypertensive later in any case. Once preeclampsia with albuminuria had developed, there was a very real danger of fœtal anoxia and resultant fœtal death *in utero*. In deciding the management of a pregnancy complicated by preeclampsia, the maternal and fœtal risks due to the preeclampsia had to be weighed against the risks of interference. At or near term, when the fœtus had attained reasonable maturity, termination of the pregnancy was indicated in any case of preeclampsia which did not completely subside with treatment. In severe preeclampsia at that stage the time to interfere was twenty-four to forty-eight hours after treatment was begun—that was at the time of maximum improvement. Severe preeclampsia before fœtal viability was not common. When it did occur, there was usually little chance of carrying on the pregnancy with the hope of obtaining a living child. Therefore, after the the usual initial treatment termination had to be considered. The greatest difficulties in management arose with the in-between group from about thirty to thirty-five weeks' gestation. Termination should not be delayed in the fulminating case, or when there was a steady deterioration in the preeclampsia. When there was definite improvement in the patient's condition and the albuminuria disappeared completely or fell to a "trace" only, it might be permissible to attempt to carry on the pregnancy to attain greater maturity of the fœtus. It had always to be borne in mind that fœtal

anoxia due to inefficient placental function might be a greater hazard than prematurity; when a pregnancy was carried on under those conditions there should be a daily review of the situation, and the pregnancy should be terminated when further conservative treatment appeared unwarranted.

Referring to methods of termination of pregnancy, Dr. Long said that induction of labour by medicinal stimulation was most unreliable before term. Surgical induction by artificial rupture of the membranes was in general the method of choice. If labour had not commenced by the following day, medicinal stimulation might be tried. Although Cæsarean section was a rapid and certain method of terminating pregnancy, there were several serious disadvantages. From the mother's point of view it was a major surgical operation and therefore carried a definite risk. A scar in the uterus created its own problems in future pregnancies. Cæsarean section did not guarantee a live child. Cæsarean section was the method of choice if another obstetrical indication was present. When labour had not commenced after artificial rupture of the membranes and the preeclampsia was not under control, or when fœtal distress was diagnosed and the conditions for vaginal delivery were not present, Cæsarean section had to be considered. The place of Cæsarean section in cases of fulminating preeclampsia was still a subject for debate. Perhaps the nearest approach to an answer at present was that if the condition could be held at bay whilst vaginal delivery was achieved, then the patient would be much better off. If termination was necessary before viability had been reached, abdominal hysterotomy was the method most often employed. With the intravenous drip administration of "Pitocin", usually after artificial rupture of the membranes, most of the patients could be delivered vaginally. Unfortunately the intravenous drip administration of "Pitocin" was not without serious risks, and it should never be employed except in a hospital with a competent resident staff constantly in attendance. Those conditions did not apply to the bulk of the midwifery practice in Australia.

Dr. Long said, in conclusion, that with conscientious antenatal care and intelligent application of principles which were now established, eclampsia had become virtually a preventible disease, and the morbidity and mortality of preeclampsia were greatly reduced. Perhaps when the ætiology was finally established preeclampsia would become completely preventible.

SPECIAL GROUP OF NEUROLOGY AND PSYCHIATRY (NEW SOUTH WALES BRANCH).

THE meeting of the Special Group of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association, which was to take place on May 16, 1957, will now be held on May 13. At this meeting, which will begin at 8 p.m. in the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, papers on the subject "Recent Advances in Psychiatry Overseas" will be read by Professor W. H. Trethowan and Dr. D. C. Maddison. The alteration in the date of the meeting has been made in order to enable medical practitioners to attend the lecture on "Tranquillizing Drugs", to be given by Professor J. M. Robson on May 16, as well as the meeting of the Special Group of Neurology and Psychiatry.

VICTORIAN BRANCH NEWS.

Section of Industrial Medicine.

THE next meeting of the Section of Industrial Medicine of the Victorian Branch of the British Medical Association will take the form of a plant walk at Electronics Park, Hamilton Street, Huntingdale, at 8.15 p.m. on Tuesday, May 21, 1957. Cathode ray tube manufacture and its associated hazards will be studied. There will be a demonstration of investigational techniques presented by the Industrial Hygiene Department. All members of the British Medical Association are welcome. Inquiries may be addressed to the Honorary Secretary of the Section of Industrial Medicine, Dr. W. F. Cooper, c.o. General Motors-Holden's, Limited, Office of the Medical Director, Medical Centre, Fisherman's Bend.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

PROVISIONING THE FIRST FLEET.[1]

(Captain Phillip to Under Secretary Nepean.)

London 2 Dec 1786.

SIR,

As I fear much discontent in the garrison if there is no allowance of wine or spirits (to which they have ever been accustomed) until spruce beer can be procured for them I beg that you will please to lay this circumstance before the Lords of the Treasury.

I shall be able to receive a certain quantity of wine on board at Teneriffe or the Cape if judg'd necessary and which I hope will be the case.

I have &c.,

A. PHILLIP.

Correspondence.

THE MEDICAL PROFESSION AND THE PLIGHT OF THE ABORIGINES.

SIR: Five members of the Western Australian State Parliament spent five weeks in the Laverton-Warburton Range area investigating the native welfare conditions in this region. This select committee published their findings and the report was presented by the Chairman, Mr. W. L. Grayden, on December 12, 1956. Portion of this report should be of interest to the medical profession, in that it deals with the medical welfare of a large group of people. I can do no better than quote directly from this document:

> It is estimated that there is in the vicinity of four hundred natives in the Laverton area and a further three or four hundred or more on the Warburton Reserve. In addition, the Warburton group is augmented by another two or three hundred or so natives who periodically visit the Warburton Range from across the South Australian border. . . . A few days before the Committee reached the mission, a party of about 19 natives from the area east of the Rawlinson Range reached the mission. It was the first time that they had been into the mission and the first time that many of them had seen white people. They had arrived in a starving condition and were extremely emaciated when seen by members of the Committee. Two at least of the children, although possibly seven and nine years old, had arms which on the upper portions were no more than an inch in diameter and their thighs would be little thicker, there being practically only skin and bone at these points. One child weighed not much more than a stone and a half and the other was possibly even lighter. . . . The most prevalent trouble is trachoma, the matron of the Warburton Mission Hospital being of the opinion that the incidence of active trachoma amongst mission home and camp children was about 80 per cent. The percentage in women was about 75 per cent. and the men 65 per cent. A large number of women are blind in one eye at least and the other eye is suffering from trachoma.

The authenticity of this report was reinforced by some films that I saw taken by Mr. W. L. Grayden and Pastor Douglas Nicholls.

The medical attention received by the white population of Australia ranks with the best in the world; yet a section of our people receive perhaps the poorest medical attention in the world. We spend hours treating over-fed neurotics (and quite rightly so), whilst others in the community are suffering from malnutrition. If the life of a white citizen is threatened by fire, flood, starvation or thirst, then the Army and Air Force are called to his aid within hours (and quite rightly so); but the starving natives under

[1] From the original in the Mitchell Library, Sydney.

similar circumstances depend upon inadequate charity. A new Australian develops an appendicitis on Heard Island, and a destroyer is diverted to help him (and quite rightly so) at the cost of £40,000. This amount would go a long way towards saving the lives of innumerable aborigines.

I feel sure that if any doctor in Australia were brought face to face with these suffering natives, he would without hesitation offer his services free and willingly to help them. But our profession should not content itself with such a passive role as this; it should urgently seek out ill health wherever it be and treat it. I feel that the problem is serious enough to warrant investigation and action by the Federal Council of the British Medical Association.

At the moment, responsibility for the aborigines rests with each State, since the Australian Constitution (Section 51, Part 5, Clause 26 of 1901) expressly denies to the Federal Parliament power to make laws with respect to aborigines. In my opinion their salvation depends upon a change being made in the Constitution so that there could be created a Commonwealth Department of Native Affairs to coordinate policy and administration throughout Australia.

Whether the readers of this letter agree with this or not is unimportant; the urgent thing is for action to prevent the aborigines dying as a race and as individuals.

Yours, etc.,

BARRY E. CHRISTOPHERS.

210 The Avenue,
Parkville,
Victoria.
April 14, 1957.

TRAINING IN SURGERY.

SIR: The Australian Council of the College of General Practitioners has advised the Federal Council of the British Medical Association that it is in agreement with the proposal made in your leading article of April 13, 1957, that a conference should be convened to discuss the question of the surgical training of general practitioners.

It is possible that the way to a solution of the problem would be opened if the Royal Australasian College of Surgeons were to prescribe a course of training leading to a general practitioner qualification in surgery (say a D.R.A.C.S.), analogous to the D.Obst.R.C.O.G.

Yours, etc.,

W. A. CONOLLY,
Chairman, Australian Council,
The College of General Practitioners.

131 Macquarie Street,
Sydney,
April 23, 1957.

SURVIVAL CURVES AS A MEANS OF INVESTIGATING CANCER.

SIR: In his stimulating article in the journal of April 20, 1957, Dr. Douglas says on page 536:

> When patients are followed up it is found that the survival rates at two years, five years and ten years become progressively worse.

And again:

> The form of my curve is generally similar to that given by the Copenhagen Radium Centre, and as far as the curve continues—for nineteen years—the survival rate becomes less.

Does Dr. Douglas mean "rate" in its usually accepted sense of a quantity per unit, or only as a percentage without reference to a unit of time?

In order to study this, it is informative to graph the survival curves on log-linear paper instead of ordinary paper. On log-linear graph paper the vertical axis with the percentage of survivals is on a logarithmic instead of a linear scale. A straight line on such a graph indicates an exponential relationship, and this occurs with some of the survival curves in Figure II, "Cancer of the Breast". A log-linear graph readily allows us to compare rates of survival and of death and shows us that the nineteen year survival figure (corrected) of 22% would be better in a regularly dying group than any of the preceding percentages of 33% for ten years and 40% for five years and 64% for two years, in groups whose patients died at regular rates and produced the survival percentages. For example, all the members of a group producing 64% survivors in two years would be dead by nineteen years, and we see that the survival rates for the longer periods are better than those for the shorter periods when considered in relation to the time producing the survival figure.

The log paper simplifies comparison between various survival and death rates, because the slope is steeper with the higher death rates and flatter with high survival rates.

On the log paper the graph for the normal expectations is almost straight (within 2%), and we can say that the relationship is exponential to a considerable degree. This means that the survival and death rates are a constant proportion of the group at any point along the graph, and the survival rate of the normal expectation cases is 98% per year, with a death rate of 2% of the group per year.

The mastectomy for carcinoma of the breast cases form a straight line (within 3%) from nil to five years, with a survival rate of about 85% and a death rate of 15% of the group for each of the first five years.

The graph flattens out between five and ten years and again forms a straight line (within 3%) over the second decade from ten to nineteen years. The survival rate per year over the second decade is about 95%, with a death rate of 5% of the group each year. So any of the mastectomy cases for carcinoma in the second decade fall into a group with a survival almost as good as the normal expectation people.

Unfortunately longer survivals are not available for the series, and we do not know how long the exponential relationship holds.

The untreated derelicts of Deland in New York form a perfect exponential graph down to nine years, with a straight line from nil to nine years and a constant survival rate of 80% and death rate of 20% of the group every year.

One could not say whether the dereliction or the carcinoma caused this constant 20% death rate per year. Both probably contributed.

Exponential or logarithmic relationships occur in the absorption of X rays and decay of radioactive substance, in which coefficients can be determined and are called absorption coefficients or transformation constants. They occur in laws of organic growth, and the amount of tissue formed by regularly dividing cells bears an exponential or logarithmic relation to the amount present at an earlier time.

In the event that exponential or logarithmic relationships were significant in survival curves, their analysis may be able to indicate when a combination of factors were operating.

It may be that the total mass of cancer tissue is a factor in prognosis and is related to the form of the survival curve. If this were true, the removal of cancer tissue to eliminate quantity alone may be justified in certain cases. These are speculations suggested by study of Dr. Douglas's article, and he is to be congratulated for the critical thought that he has put into the subject and the questions he has raised.

Yours, etc.,

ALAN GRANT.

103 Ryde Road,
Pymble,
New South Wales.
April 25, 1957.

AN UNUSUAL REACTION TO A TRANQUILLIZING DRUG.

SIR: Among the many "tranquillizing" drugs which have recently been introduced, meprobamate ("Equanil" and "Miltown") appears to have found widespread acceptance by the medical profession. The public has been so educated, if that be the right word, by popular magazines and journals, that patients frequently request treatment with "tranquillizing" drugs which may, under existing regulations, be obtained without a doctor's prescription. Exposed to everyday stresses and strains, it seems natural enough that when we are offered something which "relaxes both the body and the mind", we should readily accept such a pill in the hope that man may return to a bucolic and idyllic emotional state. The chances that a particular drug will harm a particular patient are probably small, having regard to the great number of patients who are now being given these drugs. On the other hand, toxic and allergic reactions to these compounds should be reported when they occur, so

that their pharmacological action may be better understood, and more care may be exercised by selecting cases suitable for treatment. The only reaction to meprobamate which I have hitherto encountered is described below, and is similar to cases described by Gottlieb (1956) and by Friedman and Marmelzat (1956).

Meprobamate is 2-methyl-2-n-propyl-1,3-propanediol dicarbamate, and according to Berger (1954) it resembles mephenesin in producing reversible paralysis of the voluntary muscles without significantly affecting autonomic functions. It acts on the central nervous system and inhibits internuncial circuits. It is antagonistic to strychnine and counteracts symptoms from released inhibitions during light anæsthesia. It also augments anæsthesia with barbiturates. There is little direct action on the muscles, no effect at myoneural junctions, and no blocking of conduction of peripheral nerves. Closely related to mephenesin, it has a much longer duration of action and is rapidly absorbed through the gastro-intestinal tract. Approximately 10% is excreted unchanged, and the remainder conjugated with glucuronic acid in the urine.

My patient was a fifty-two year old man, suffering from considerable anxiety and depression, with tension headaches, dizziness, fatigue and listlessness. He had never before had a drug eruption, nor were there any known allergic symptoms. He took one tablet of "Equanil" (400 milligrammes) on the night of April 18, 1957, and woke during the night with marked sweating, generalized skin irritation and burning and a temperature of 101° F. In the morning there was a generalized urticarial reaction, most marked on the trunk, with patches of a maculo-papular eruption. The following morning the rash had become localized to the front of the abdomen, extending from the umbilicus to about two inches below the groins, not involving the genitals, and it was now purpuric in appearance. There was marked tenderness in the right inguinal region. Following the use of an antihistamine and sedation, the rash faded within two days. The next day, in order to establish the cause-and-effect relationship, a further tablet of "Equanil" was given. Within three hours there was intense burning and irritation of the entire skin, and recurrence of the purpuric rash, more marked than previously, in the same region, also involving the flexor aspects of both knees. The itching was marked and extreme in character. Twelve hours after the patient's ingestion of the pill, the eruption began to clear, and had disappeared completely after a further two days.

It would be interesting to know what explanations may be offered for the unusual site of predilection in this case.

Yours, etc.,

187 Macquarie Street, Sydney,
April 26, 1957.

J. SCHNEEWEISS.

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Obituary.

DUDLEY WILLIAM CARMALT JONES.

PROFESSOR DUDLEY WILLIAM CARMALT JONES died recently in England at the age of eighty-two years. Born and educated in England, Professor Carmalt Jones had already a distinguished professional and service record before he went out to New Zealand in 1920 to take the Chair of Systematic Medicine in the University of Otago. Despite some initial difficulties he remained in this position for nearly twenty years and made a distinguished contribution to medicine and medical education in New Zealand. On his retirement from the chair he was made Emeritus Professor of Systematic Medicine in recognition of his services. He was President of the New Zealand Branch of the British Medical Association in 1935 and was a foundation Fellow and Vice-President of The Royal Australasian College of Physicians.

A former student, D.R., writes: Carmalt Jones was beloved by all, who recognized in him the scholar and gentleman in addition to the physician. Maybe he was a little "old school", but he strove to keep us abreast of developments. He was fine company, and lost no chance of introducing us to Plato and the Greeks through Sir Richard Livingstone's little books. His own history of the Otago School is a valued bequest. Those who were fortunate to meet him latterly in his retirement near London found him the same kindly interested "C.J." of thirty years ago in Dunedin.

MARY BOYD BURFITT WILLIAMS.

We are indebted to Dr. Marjory Little for the following appreciation of the late Dr. Mary Burfitt Williams.

Mary Boyd Burfitt Williams, who died on November 30, 1956, belonged to a medical family, her two brothers, husband and three sons all being members of the medical profession. She was educated at Saint Mary's College, "Rosebank", by Good Samaritan nuns, and proceeded to the University of Sydney, where she graduated in arts, science and medicine.

At the end of her first year in medicine she gained Professor Haswell's prize for zoology and shared the Renwick Scholarship for general proficiency. She graduated in 1909 with first-class honours, and was appointed to the Royal Prince Alfred Hospital as a resident medical officer, sharing with the late Dr. Elsie Dalyell the honour of following Dr. Jessie Aspinall, who was the first woman to gain this appointment.

At the end of her junior year, Mary Burfitt Williams was invited to remain on the staff as a senior resident medical officer—the first woman to gain this recognition of good work. She was also the first woman to be appointed a resident medical officer at the Women's Hospital, Crown Street. She joined the staff of Lewisham Hospital in 1912 as an honorary physician, and was responsible for the establishment of the department of pathology in that hospital. She remained an active member of the honorary medical staff till 1938.

Mary Burfitt Williams carried on a successful general practice at Glebe Point from 1912 to 1924, and from 1924 to 1952 she practised in Macquarie Street as a consulting physician. She was a councillor of Sancta Sophia College within the University of Sydney from its establishment in

1929 until 1953, when she resigned, as she felt her place should be taken by a younger person.

Mary Burfitt Williams was one of the pioneer medical women of the University of Sydney to whom younger medical women will always be indebted, for these pioneer women never forgot how much the future of all medical women depended on the success they themselves achieved in their pioneering efforts. She was a careful, sound physician, giving her patients confidence and gaining their affection and respect. She was a devoted wife and mother, and a loving, lovable friend.

ISAAC JONES.

ISAAC JONES, who died in London on March 24, 1957, was an Australian who retained a strong link with Australia although he had made England his home. Born in Melbourne in 1882, he studied medicine at the University of Melbourne, from which he graduated in 1909. A doctorate in medicine with honours followed in 1912. He served on the staff of the Melbourne Hospital and the Women's Hospital before he joined the Royal Army Medical Corps in the first World War. He did not return to Australia on demobilization, but was admitted to membership of the Royal College of Physicians in 1919 and commenced a long association with Saint Thomas's Hospital. He was subsequently elected a Fellow of the Royal College of Physicians, and amongst his more notable appointments was that of Chief Medical Officer and Consultant Physician to the Metropolitan Police, Scotland Yard. For some twenty years he represented the New South Wales and Queensland Branches of the British Medical Association on the Central Council of the Association. He leaves a widow and two sons, to whom we extend our sympathy.

University Intelligence.

THE UNIVERSITY OF WESTERN AUSTRALIA.

THE following information has been taken from the *University Gazette*, University of Western Australia.

The Medical School.

On February 4, 1957, 14 sixth year medical students from the University of Adelaide enrolled for the final year course at the School of Medicine, Victoria Square, Perth. The Vice-Chancellor and the Dean, Professor Gordon King, briefly addressed the students, who also met members of the medical professoriate. Studies began on February 5, the students being grouped and attached to medicine, surgery and obstetrics and gynæcology. Teaching is carried out in the School of Medicine, the Royal Perth Hospital, the King Edward Memorial Hospital and the Princess Margaret Hospital for Children, and visits will be made to other specialist hospitals. At the end of 1957, those who pass the final examinations will be entitled to the degrees of Bachelor of Medicine and Bachelor of Surgery of the University of Adelaide.

The applications for enrolment for the first year in 1957 are approximately 90; this figure shows an increase of 32 over 1956.

In 1958, first, second, fifth and sixth year courses will be conducted, and in 1959 the whole course will be offered in the University. Those who complete the final examination in 1959 will be the first to graduate M.B., B.S. from the University of Western Australia.

The following appointments have been made in the School of Medicine:

Department of Medicine.—Senior Assistant (temporary): R. B. Lefroy, M.A. (Oxford), M.B., B.S. (Melbourne), M.R.A.C.P.; Junior Assistant (temporary): P. E. Hurst, M.B., B.S. (Adelaide); Social Worker (temporary): Miss F. Pitman, Dip.Soc.Stud. (Melbourne).

Department of Surgery.—Senior Assistant (temporary): N. Way, F.R.C.S., F.R.A.C.S.; Junior Assistant (temporary): R. Paton, M.B., B.S. (Adelaide), F.R.C.S., F.R.C.S. (Edinburgh).

Department of Obstetrics and Gynæcology.—Senior Assistant (temporary): A. G. Mathew, M.B., B.S. (Melbourne), M.R.C.O.G.

Department of Pathology.—Senior Lecturer (temporary): J. H. Little, M.B., B.S. (Melbourne), D.P.H. (Sydney), D.C.P. (London); Medical Illustrator: H. Upenieks.

Visiting Lecturers.—Psychological Medicine: Francis Michael Gerald Prendergast, M.B., B.S. (Melbourne), D.Psy. (Edinburgh); Otology: Ernest Joseph Green, M.B., B.S. (Melbourne), D.L.O. (Melbourne), F.R.A.C.S.; Tuberculosis: Alan Joseph King, B.Sc., M.B., B.S. (Melbourne), F.C.C.P.; Anæsthetics: James Miller Saunders, M.B., Ch.B., B.A., M.F.A., R.A.C.S.; Diabetes: Bruce Atlee Hunt, M.D., B.S. (Melbourne), F.R.A.C.P., F.R.C.P.

Royal Australasian College of Surgeons.

SIMS COMMONWEALTH TRAVELLING PROFESSOR.

SIR JAMES PATERSON ROSS, the Sims Commonwealth Travelling Professor for 1957, will be visiting Sydney in May, 1957. His programme will be as follows: Monday, May 13: 9.30 a.m. to 12.30 p.m., visit to Saint Vincent's Hospital. Tuesday, May 14: 9.30 a.m. to 12.30 p.m., visit to the Royal North Shore Hospital; 8.15 p.m., lecture at the Stawell Hall, 145 Macquarie Street, "The Changing Scope of Surgery". Wednesday, May 15: 11.15 a.m., visit to Sydney Hospital. Friday, May 17: 2 p.m., visit to Royal Prince Alfred Hospital; 8.15 p.m., lecture at the Stawell Hall, "Inflammatory and Idiopathic Œdema". These meetings are open to all medical practitioners.

ALAN NEWTON PRIZE.

IN 1951 the sum of £1042 was subscribed to provide a prize to recognize the services to the Royal Australasian College of Surgeons of Sir Hibbert Alan Stephen Newton, Kt., a Foundation Fellow and, later, a President of the College. This sum of money has been invested in authorized trustee investments and the interest used to provide a prize for essays on practical surgical subjects.

The prize is to be awarded under the following conditions:

1. The Alan Newton Prize shall be awarded biennially.

2. Candidates for the prize shall be Fellows of the Royal Australasian College of Surgeons (not being members of the Council), the Royal College of Surgeons of England, the Royal College of Surgeons of Edinburgh, the Royal College of Surgeons in Ireland or Fellows in Surgery of the Royal Faculty of Physicians and Surgeons of Glasgow.

3. Essays must be typewritten in English and not to exceed 75,000 words. Case histories of cited cases must not be included in the typescript but placed after it in an appendix.

4. Each essay must be distinguished by a motto and accompanied by a sealed envelope containing the name and address of the author and having on the outside of the envelope the motto corresponding to that on the essay.

5. Essays must reach the secretary on or before December 1 in the appropriate year.

6. The prize essay and accompanying illustrations and preparations will become the property of the College.

7. Authors may claim essays not awarded prizes upon authenticated application within two years.

8. If no essay is adjudged worthy of the prize no award shall be made.

9. Any unexpended interest may be added to the principal of the fund.

The subject for the next essay will be "The Surgery of Malformations of the Heart and Great Vessels". All entries must be in the hands of the secretary of the College on or before December 1, 1958.

Award for 1956.

The Council of the Royal Australasian College of Surgeons has awarded the Alan Newton Prize for 1956 to Mr. L. J. Parton, F.R.C.S., of Auckland, New Zealand. Mr. G. R. Davidson, F.R.C.S. (Edinburgh), F.R.A.C.S., was *proxime accessit*.

The World Medical Association.

OFFICERS AND OFFICIALS.

THE current officers of The World Medical Association are as follows: *President*, Dr. J. A. Bustamante (Cuba); *President-Elect*, Dr. Ahmet Rasim Onat (Turkey); *Treasurer*, Dr. Ernst Fromm (Germany). Council officials include the following: *Chairman*, Dr. Lorenzo Garcia-Tornel (Spain); *Assistant to Chairman*, Dr. Leon Gerin-Lajoie (Canada); *Consultant-General*, Dr. T. C. Routley (Canada); *Executive Editor*, Dr. Austin Smith (United States of America); *Associate Editor*, Dr. S. S. B. Gilder (Canada); *Official Liaison Officer*, Dr. Jean Maystre (Switzerland); *Secretary-General*, Dr. Louis H. Bauer (United States of America).

29TH COUNCIL SESSION.

THE Norwegian Medical Association will be host to the 29th Session of the Council of The World Medical Association in Oslo, Norway, from April 29 to May 5, 1957.

The Council is the executive body of The World Medical Association, and includes the elected officers, Council members and officials of the organization. Twenty-three members from fifteen countries are expected to attend, one each coming from as far away as Australia, India and Chile.

Agenda.

The Council at this session prepares its reports and recommendations for the 11th General Assembly, to be held in Istanbul, Turkey, from September 29 to October 5, 1957. The following are some of the topics under consideration: (i) Ways and means of implementing world-wide recognition of and assuring protective powers to the emblem for civilian doctors and their units in time of war. (ii) International medical law. (iii) The trend in some countries toward establishing various "degrees" of medical qualification. (iv) Medical secrecy under medical social security plans. (v) The programme of the Second World Conference on Medical Education (Chicago, 1959). (vi) Conclusions and recommendations resulting from the First Latin American Hospital Conference (Havana, 1956). (vii) The role of the Regional Secretary and Secretariat. (viii) Priority in expenditure of association funds. (ix) Central repository for medical credentials—progress report. (x) Desirability of compiling and publishing reports on the conflicts between medical organizations and various government agencies in certain countries, and the outcome of such conflicts. (xi) Liaison officers' reports on activities of other organizations of interest to the World Medical Association. (xi) Conditions of medical practice in Cuba and in Norway.

Representatives to World Health Assembly.

After the close of the 29th Council Session of The World Medical Association, some of its members will go to Geneva, Switzerland, to attend the 10th World Health Assembly of the World Health Organization.

Research.

THE AUSTRALIAN NATIONAL UNIVERSITY: THE JOHN CURTIN SCHOOL OF MEDICAL RESEARCH.

APPLICATIONS are invited for scholarships in the John Curtin School of Medical Research. Applicants should have one of the following qualifications: M.B., B.S., M.Sc., B.Sc. (honours) or their equivalent. Scholars are expected to enrol for a Ph.D. degree, and will normally be expected to commence their duties in September, 1957. The scholarships are tenable for two years and may be renewed for a third year.

Allowances are as follows: £A771 net *per annum* if single; additional allowance usually granted to a married scholar with children; grant towards scholar's travel to Canberra.

The fields of research in which scholarships are offered, and the senior investigators in each field, are as follows:

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 20, 1957.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	2(2)	2	..	..	..	..	..	5
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	8	..	..	..	..	..	..	..	8
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	2(1)	..	..	..	..	..	..	..	2
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	3	..	..	..	..	..	1	..	4
Diphtheria	1	..	..	..	..	2	..	..	3
Dysentery (Bacillary)	..	2(2)	3(2)	3(2)	..	..	..	..	8
Encephalitis	1	..	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	33(13)	26(13)	..	8(3)	1(1)	..	2	..	70
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	1	..	3	..	..	..	..	..	4
Malaria	..	..	1	..	..	..	1	..	2
Meningococcal Infection	1	1(1)	..	..	..	..	..	..	2
Ophthalmia	..	..	..	2	2	..	1	..	5
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	1(1)	..	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	1	1	1	..	..	..	..	..	3
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	13(11)	..	9(3)	2(2)	..	..	..	24
Salmonella Infection	..	..	..	1(1)	1(1)	..	..	..	2
Scarlet Fever	8(4)	13(8)	2	6(4)	2(2)	..	..	..	31
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	2(1)	..	..	..	2
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	33(17)	19(13)	8(3)	2(2)	12(5)	2	..	..	76
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	4(4)	..	..	..	..	..	4
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Biochemistry: Professor A. H. Ennor and Dr. J. F. Morrison—biochemical studies on phosphorylated guanidines; Dr. R. L. Blakley—relation of *B* group vitamins to biosynthesis; Dr. F. D. Collins—biochemistry of phospholipids.

Experimental pathology: Dr. G. B. Mackaness—hypertension and atherosclerosis.

Medical chemistry: Professor A. Albert and Dr. D. J. Brown—heterocyclic chemistry.

Microbiology: Professor F. J. Fenner—field and laboratory studies of myxomatosis; genetic studies of pox viruses; Dr. S. Fazekas de St. Groth and Dr. H. J. F. Cairns—quantitative studies on the multiplication of influenza virus; Dr. W. K. Joklik—biochemical studies of virus multiplication.

Physiology: Professor J. C. Eccles and Mr. J. S. Coombs—biophysical studies on nerve cells; synaptic and neuromuscular transmission; Dr. D. R. Curtis—studies on pharmacology and synaptic transmitter substances in the central nervous system.

Application forms and other particulars may be obtained from the Registrar, Australian National University, G.P.O. Box 4, Canberra, A.C.T., to whom applications should be submitted by May 31, 1957.

Notice.

ILEOSTOMY ASSOCIATION.

A NUMBER of people in Victoria who have ileostomies have arranged to meet in Melbourne on Saturday, May 18, at 2 p.m. At this meeting an ileostomy association will be formed. The object of the association is to link up with similar organizations in the United States and England, and so facilitate the welfare of its members. Further information may be obtained by telephoning JL 4113 or JM 1482 (Melbourne).

SIR HAROLD DEW COMMEMORATION FUND.

A FUND has been established to commemorate the service to the School of Medicine in the University of Sydney given by Sir Harold Dew, who has retired from the Chair of Surgery. The committee appointed to administer the fund is anxious that all Sydney medical graduates should have an opportunity to contribute; to this end it has proposed that the maximum donation should be £5 5s. The chief aim of the fund is to convert a room in the Department of Surgery within the New Medical School into the "Sir Harold Dew Study Room", to be equipped as a reading and conference room for graduates and surgical trainees. The fund will also be used in other ways to advance postgraduate work within the Department of Surgery; to this end three trustees have been appointed to administer it—the Dean of the Faculty of Medicine, the Professor of Surgery and Sir Norman McAlister Gregg. The Senate has approved the fund, and donations may be charged as income tax deductions. Contributions should be sent to the Honorary Treasurer, University of Sydney Harold Dew Fund, Department of Surgery, University of Sydney, Sydney. Cheques should be crossed and made payable to "The University of Sydney Harold Dew Fund".

Deaths.

THE following deaths have been announced:

JAMES.—Stanley George James, on April 11, 1957, at Crowthorne, Berkshire, England.

STEPHEN.—Clive Travers Stephen, on April 17, 1957, at Melbourne.

GINSBERG.—Maurice William Ginsberg, on April 22, 1957, at Sydney.

FORSYTH.—Robert Leslie Forsyth, on April 26, 1957, at Surrey Hills, Victoria.

POWYS.—Norman Skelton Powys, on April 29, 1957, at Sydney.

THOMPSON.—George Alexander Thompson, on April 29, 1957, at Sydney.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Phipps, Gilbert William, M.B., B.Chir., 1955 (Univ. Cambridge), c.o. Bank of New South Wales, 341 George Street, Sydney.

Medical Appointments.

Dr. C. H. C. Searby has been appointed President of the Medical Board of Victoria, pursuant to the provisions of Section 3 of the *Medical Act*, 1928, of Victoria.

Dr. J. R. de Vos Toussaint has been appointed Medical Officer, Mental Hygiene Branch, Department of Health, Victoria.

Dr. Pauline M. Daniels has been appointed Assistant Director of Anæsthesia at the Royal Adelaide Hospital.

Diary for the Month.

MAY 13.—Victorian Branch, B.M.A.: Finance Subcommittee.
MAY 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 15.—Western Australian Branch, B.M.A.: General Meeting.
MAY 16.—Victorian Branch, B.M.A.: Executive of Branch Council.
MAY 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 22.—Victorian Branch, B.M.A.: Branch Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 *per annum* within America and foreign countries, payable in advance.

ILLUSTRATIONS TO THE ARTICLE BY K. VINER SMITH.

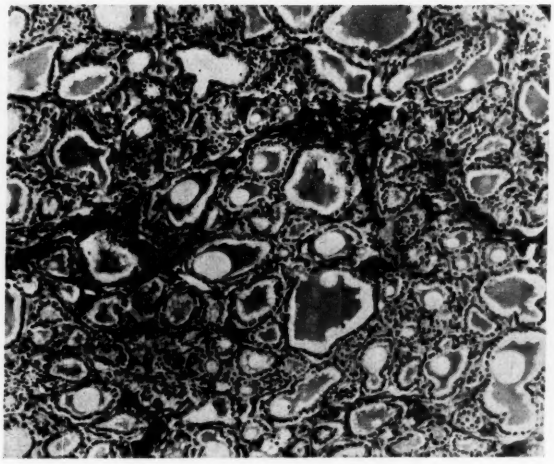

FIGURE I.
Normal thyroid (×90) from a child, aged one year.

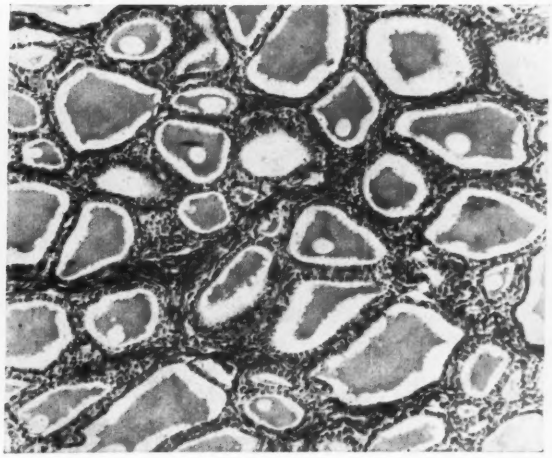

FIGURE II.
Normal thyroid (×90) from a child, aged ten years. The vesicles are larger than in Figure I.

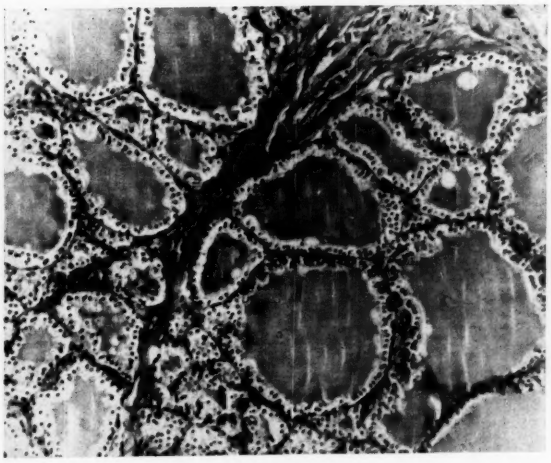

FIGURE III.
Normal thyroid (×90) from a girl, aged seventeen years. The vesicles are larger than in Figures I and II and there is greater variation in size.

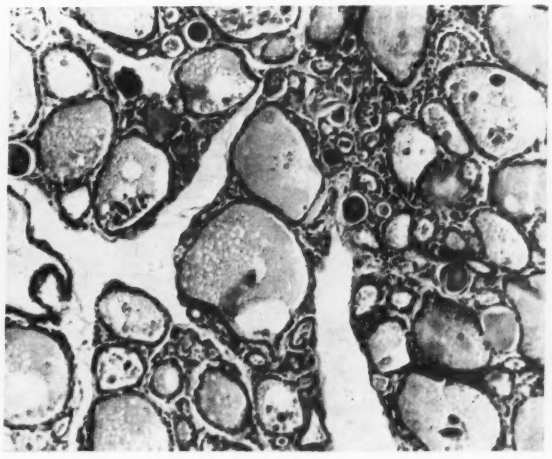

FIGURE IV.
Normal thyroid (×90) from a man, aged eighty years. There is great variation in the size of the vesicles. The larger ones are lined by flattened cells. There are many very small follicles.

ILLUSTRATIONS TO THE ARTICLE BY K. VINER SMITH.

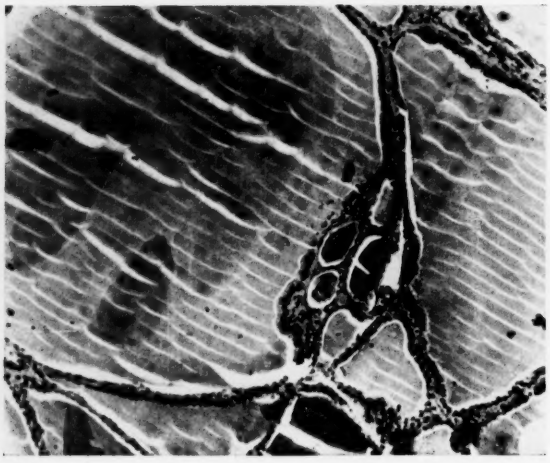

FIGURE V.
Diffuse colloid goitre (× 90). The distended vesicles are lined by flattened cells, except at one place where there is a group of tall cells projecting into the lumen.

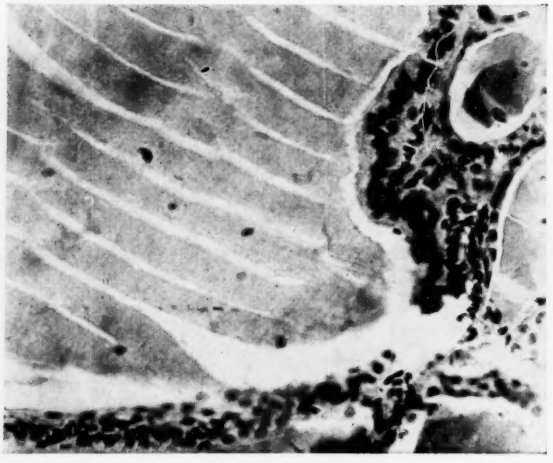

FIGURE VI.
Diffuse colloid goitre (× 320). A group of tall cells more highly magnified.

ILLUSTRATIONS TO THE ARTICLE BY A. C. L. CLARK.

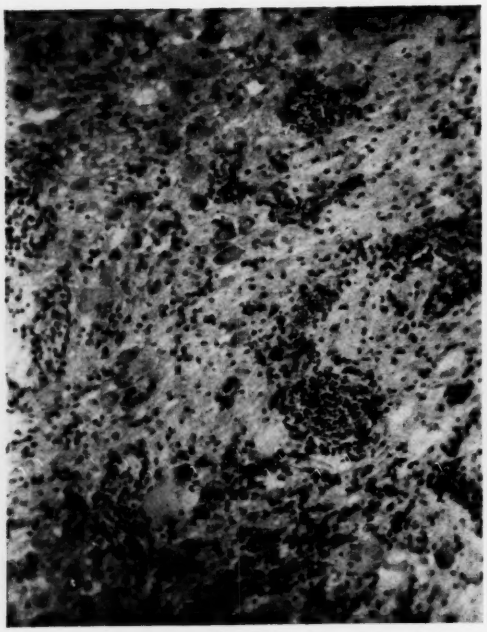

FIGURE II.
Histological appearance of tumour showing presence of numerous large cells and aggregates of small round cells. (Hæmatoxylin and eosin stain, × 100.)

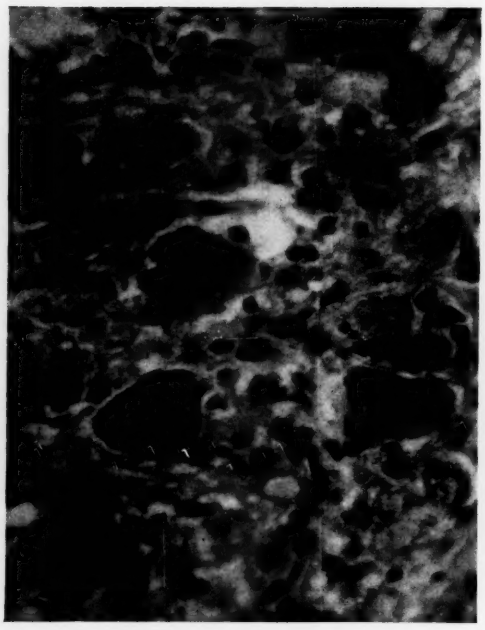

FIGURE III.
Higher power view of section of tumour showing typical nerve cells. (Hæmatoxylin and eosin stain, × 480.)

ILLUSTRATIONS TO THE ARTICLE BY P. R. HODGE AND J. R. LAWRENCE.

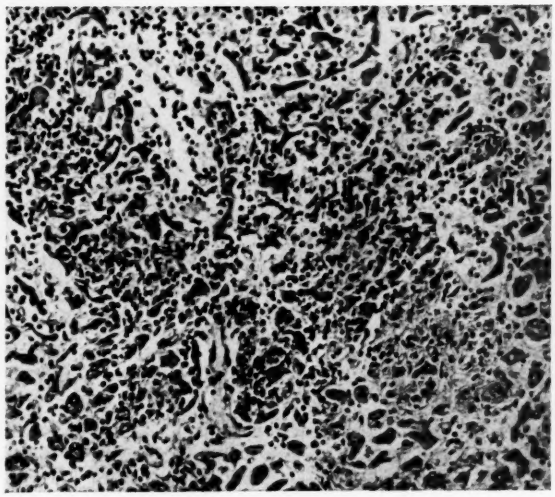

FIGURE III.

Case I: Photomicrograph showing diffuse interstitial myocarditis with extensive infiltration of muscle and degeneration of muscle fibres (×160).

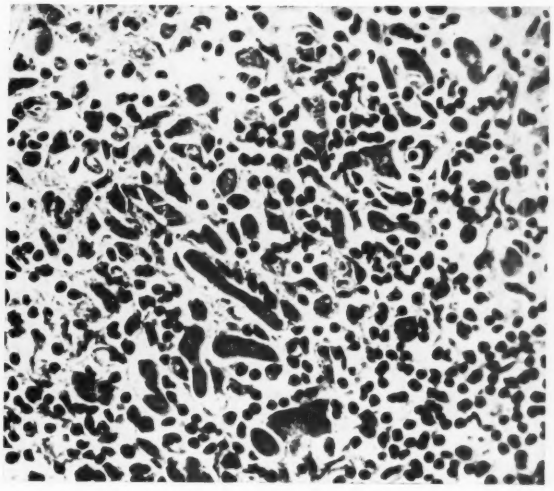

FIGURE IV.

Case I: Inflammatory infiltrate in myocardium (×500).

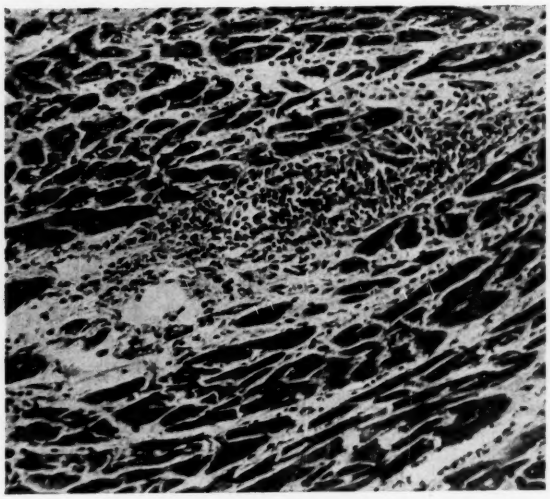

FIGURE V.

Case II: Inflammatory cells between muscle bundles in the heart (×150).

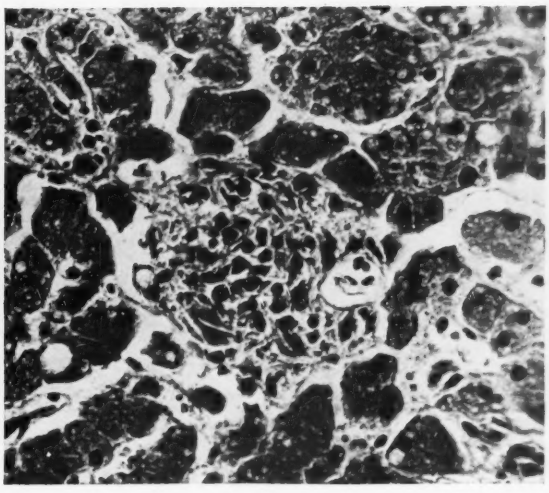

FIGURE VI.

Case II: Focal granuloma in the liver (×500).

ILLUSTRATION TO THE ARTICLE BY G. T. ARCHER, M.B., B.S.

FIGURE I.
A sickle-cell preparation of the patient's red cells.

ILLUSTRATIONS TO THE ARTICLE BY F. V. STONHAM.

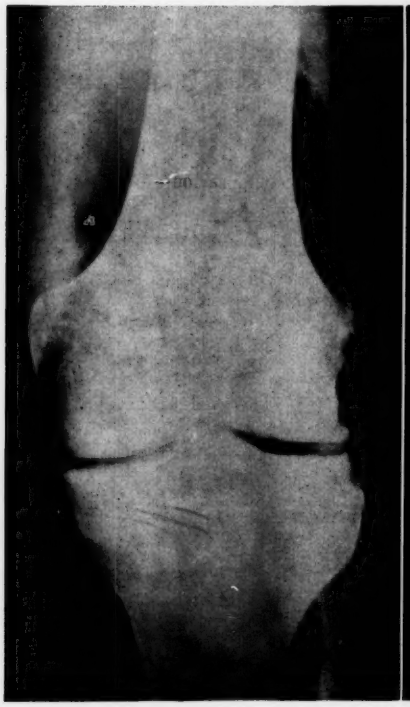

FIGURE I.
Pneumarthrogram showing incomplete previous removal of lateral meniscus.

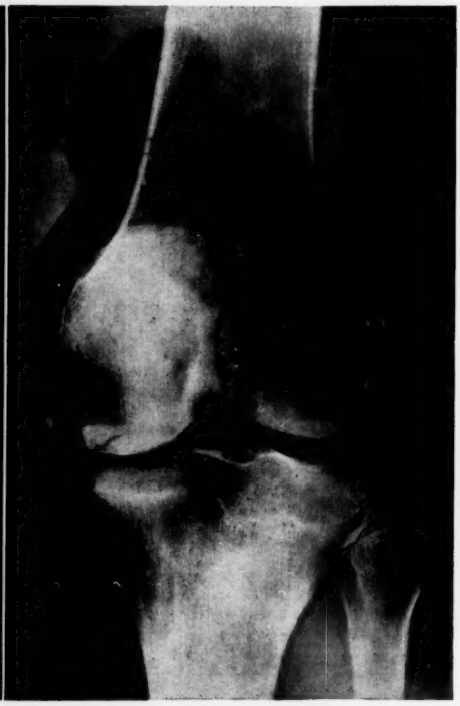

FIGURE II.
Pneumarthrogram showing peripheral tear of medial meniscus.